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THE
# LARYNGOSCOPE.

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## THE ORGAN OF CORTI BY PHASE CONTRAST MICROSCOPY.*

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### INTRODUCTION.

In studies[1-4] on the histologic effects of intense sound on the inner ear, the degree of injury and its extent within the cochlea has been described in preparations made in the usual manner. The methods of fixation, imbedding in celloidon, sectioning and staining, have served to reveal many alterations in hair and supporting cells of the organ of Corti. They have left much to be desired in the interpretation of the finer changes in the nucleus and cytoplasm of individual cells. In view of the newer and improved methods for physiologic testing it is important to know whether the organ of Corti can recover from mild injuries or to what extent its recovery might take place.

It has been necessary to study by means of the phase contrast microscope freshly isolated pieces of the organ of Corti

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†On leave of absence from Tokyo Medical and Dental University for study at Central Institute for the Deaf.

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from control animals before attempting similar observations on animals subjected to intense sound. The present results are concerned with the normal cells and are similar to the studies recently reported by Hilding[5] for the tectorial membrane. A future study will deal with the appearance of the injured cells of the organ of Corti with phase contrast microscopy.

## MATERIALS AND METHODS.

Twelve guinea pigs of about 250 to 400 gm. in body weight were used in this study. Each animal was anesthetized by an intraperitoneal injection of veterinary nembutal and finally given a second dose sufficient to kill. A temporal bone was then removed and the bulla opened to expose the cochlea. Bone adjacent to the cochlea was removed, and the specimen was then immersed in warmed physiological saline solution. Further dissection and preparation of the slides was carried out on a warm table at 37° C. The light source was a Universal microscope lamp, for which the intensity was adjusted and the beam of light focused on the specimen. A binocular dissecting microscope was used with a magnification of about 27 times.

The bone was removed from the apex and into the first turn of the cochlea with a small, sharp hook. Markings of the stria vascularis, Reissner's membrane and the osseous spiral lamina were visible as landmarks. Using small scissors, a cut was made into the modiolus and another through the stria vascularis. The piece was removed to a warm slide and further dissected in saline solution under the same magnification. A small segment of the organ of Corti was removed from the basilar membrane. This usually measured about 1 to 2 mm. in length. It was then transferred to another slide, a drop of saline solution added, and the preparation was then covered with a No. 0 coverglass and ringed with vaseline. The slide was then transferred to the stage of a phase contrast microscope and observed by means of dark, medium objectives. It was at first studied under low power magnification in order to find the areas thin enough for study and

photography. Higher magnification was used for study of single cells and most of the photomicrographs. The preparations showed no signs of deterioration before 30 to 40 minutes.

## FINDINGS.

*The Preparation:* As shown in Fig. 1, the preparation under low power magnification (108×) usually consisted of a few millimeters of the organ of Corti removed from the basilar membrane. Other preparations consisted of special mounts of the tectorial membrane, basilar membrane or preparations that included the osseous spiral lamina, organ of Corti, and spiral ligament. For most observations the short segments of hair and supporting cells held in place by the reticular membrane were used. In these the thinner areas about the periphery of the preparation were used for the study of single cells. In Fig. 1 the free margin contains external hair cells and supporting cells, and the tops of pillar cells appear as a fairly homogenous row separating the external from the internal hair cells. Attempts to tease or further isolate single cells for study proved disastrous for the sensory cells. After such treatment the reticular membrane with attached pillar cells and tops of hair and supporting cells usually remained intact. The organ of Corti can readily be lifted from the basilar membrane, but the most resistant and tenacious part of it appears to be the reticular membrane.

Attempts were made to keep the slides in a warm chamber during the time of dissection, preparation and observation. It was found that most preparations were suitable for study for a period of approximately 30 to 40 minutes, and use of the warm chamber during the period of observation did not seem to lengthen this period materially. Beginning disintegration in these fresh preparations was evidenced by: *1.* a swelling of some cells; *2.* a cessation of Brownian movement for most intracellular particles; *3.* a rearrangement of cytoplasmic constituents and the formation of a semisolid phase. This resulted in the formation of networks and a congealed appearance of the cytoplasm with *4.* formation of cytoplasmic

vacuoles. Additional changes are described under descriptions of the different cell types that comprise the organ of Corti.

*The External Hair Cells:* A single external hair cell (1,000×) is fully shown in Fig. 2. The shape is cylindrical with a nucleus in the lower one-third of the cell. The distortion of the cell in its middle one-third was found to be a common appearance for many of the hair cells in these preparations. There was very little tendency for the cells to change in shape, either in diameter or in length, during the time of observation or in old preparations. The cell membrane at all times seemed to impart some rigidity to the cell. The most basal portion was occupied by a collection of highly refractile granules (Retzius body), as seen in Fig. 4. Between the nucleus and the Retzius body the cytoplasm seemed relatively dense and granular. Just above the nucleus a collection of small nonrefractile rods (probably mitochondria) occur. They were not found dispersed through the cytoplasm as usually described in specially prepared sections of the organ of Corti. The middle one-third of the cell contained a relatively clear cytoplasm with an occasional granule showing Brownian movement. The upper one-third contained a clear background with relatively few granules but considerable substance of a dark, patchy material. Hensen's body, as described, was not seen. It is possible that the patchy material might contribute to the formation of a Hensen's body in fixed preparations. The uppermost portion of the cell appeared as a yellowish, refractile and homogenous substance from which the hairs emerged.

*The Internal Hair Cells:* The flask-like shape of the internal hair cell (1,000×) is seen in Fig. 3. The body of the cell is spherical with a narrow and short neck-like portion. The cytoplasm contains some granules distributed throughout the cell in small groups. In the base are located fewer refractile granules than in the external hair cell. They resemble in every way the constituents of the external hair cell, but they are fewer in number. The nucleus is not located centrally in the spherical body but is somewhat closer to the base of the

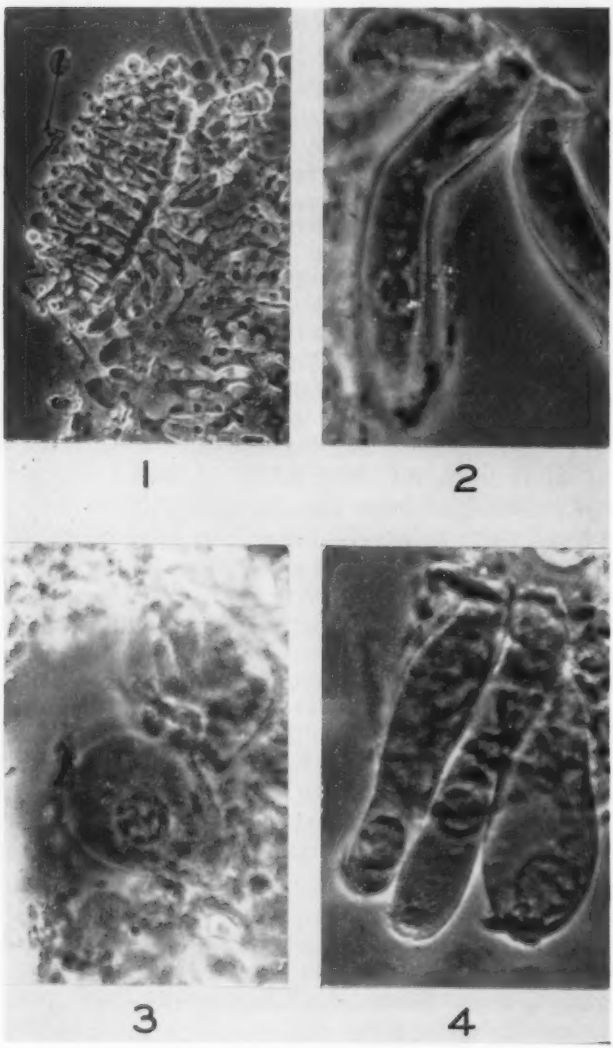
cell. A similar zone of homogenous substance through which the hairs emerge is found at the uppermost portion of the cell.

Fig. 4 offers an opportunity to compare the two types of hair cells (1,000×). The detail of the upper portions of the cells is somewhat obscured by the fibrillar processes of the pillar cells in an irregular arrangement. The nucleus of one external hair cell is located closer to the center of the cell than usual.

The inverted V-shaped pattern of the hairs as they emerge from the external hair cell is shown in Fig. 5 (1,000×). This row of cells as seen from the reticular membrane is the one closest to the external pillar rods; the more peripheral rows of sensory cells are not in focus in this photomicrograph. The hairs of the internal hair cell emerge in a short, straight line and, like the hairs of the external hair cell, each configuration is made up of several rows of hairs.

Fig. 6 is a view of the reticular membrane (2,000×) as seen from above. Its components are octagonal. In the center of the field is an external hair cell with projecting hairs that measure 5 to 8 microns in length. A lateral view (2,000×) of similar cells is shown in Fig. 7. Their length and number for each cell can be well appreciated. The plane of focus in this preparation was on the upper end of the hair cells and as a result they are not fully shown in the photomicrograph. The tectorial membrane was lifted off of the organ of Corti during dissection of the specimen so that its relations to the hairs was not observed. Since the hairs became more distinct in older preparations that had been under observation for some time, it is possible that any binding substances may have dissolved with separation of the individual hairs.

*Supporting Cells:* A Deiter's cell (1,000×) is shown in Fig. 8. It has relatively clear cytoplasm with well defined boundaries. It is a pear-shaped cell from which two fibrillar processes emerge. One process passes to the reticular membrane, another forms a cup-shaped base for an external hair cell. The fibrils originate in the cytoplasm as shown in Fig. 8.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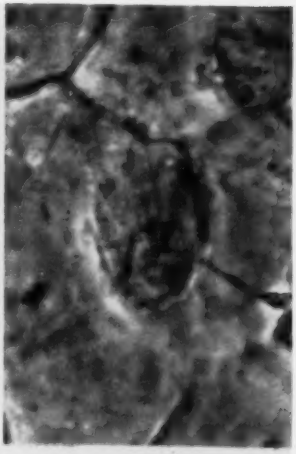

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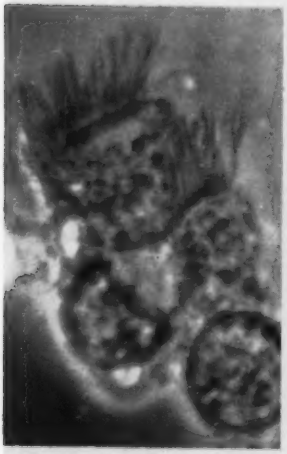

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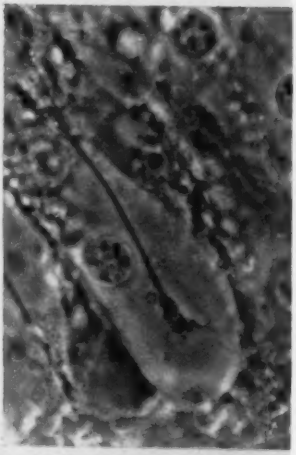

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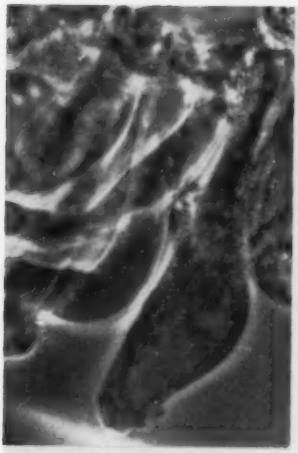

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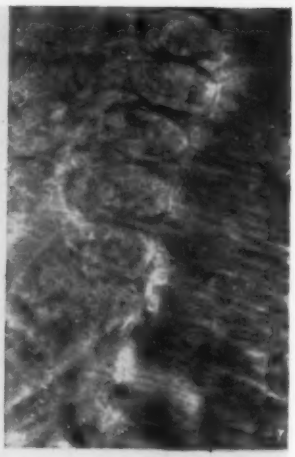

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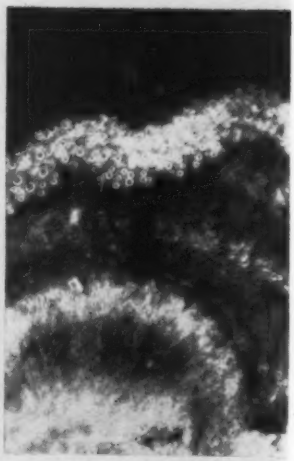

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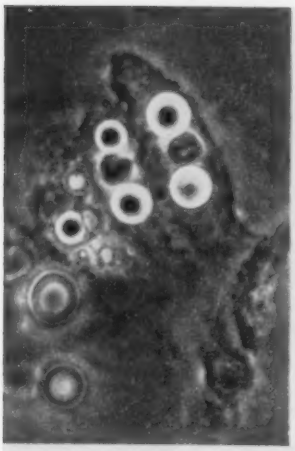

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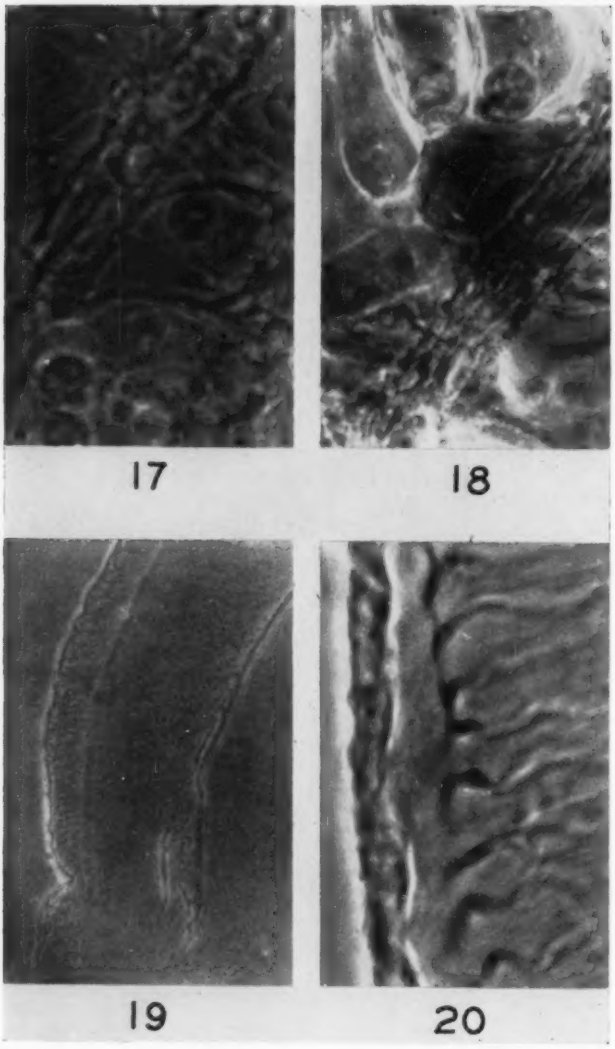
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In this instance a few granules adhere to them at their points of origin. They are usually seen to fan out into three or more strands at their origin, which is located below and to one side of the nucleus. In Fig. 9 (1,000×) are shown the two processes: the one to the left passes upwards to the base of an external hair cell, while the somewhat larger one terminates in the reticular membrane. In Fig. 10 (1,000×) the ends of fibrils can be seen between the hair cells. They are more in evidence to the right in the photomicrograph.

An external pillar cell (1,000×) with its fibrillar supporting process is shown in Fig. 11. It lies between two Deiter's cells. The nucleus is to be seen in the base of the cell that has become detached from the basilar membrane. In contrast to the fibrils in the processes of a Deiter's cell these are more numerous, and each fibril is less distinct in outline; they do not appear to have the same bristle-like rigidity. The fibrils originate in the base of the pillar cells and to one side of the nucleus. As they pass into the rod portion of the cells they become more compact and finally at the top of the tunnel form characteristic terminations that become the apex of the tunnel of Corti.

A photomicrograph of a dark field preparation (108×) of the osseous spiral lamina, basilar membrane and organ of Corti is shown in Fig. 12. The strip containing refractile globules or vacuoles represents the location of the cells of Hensen. They appear similar in preparations made from all turns of the cochlea. Under ordinary illumination they are not apparent in the fresh preparation. Fig. 13 is a higher power (1,000×) of individual Hensen's cells as observed with the phase contrast microscope. The highly refractile bodies are of varying sizes with smaller ones located usually in the tapered base of the cell. The larger ones are usually located about the middle or apex of the cell. Their significance and nature remain obscure. Fat globules have been described as occurring in the cells of Hensen in the apical turn. In fixed and stained preparations they appear as vacuoles and are restricted to the apical areas of the organ of Corti. It seems

doubtful that all of these vacuoles seen under the phase contrast microscope are fat. Further studies are under way to determine their nature by histochemical methods.

Hensen's cells are easily removed from the organ of Corti as a continuous piece. They do not seem to be anchored securely in any way to the reticular membrane and, for that reason, one wonders how much, if any, they contribute to support of the organ of Corti.

*Basilar Membrane:* The fibres of the basilar membrane (1,000×) after removal of the organ of Corti are shown in Fig. 14. This represents the appearance of the basilar membrane as viewed from above in its extent through the pectinate and arcuate zones. The fibres have a somewhat wavy appearance under this magnification, and intervening spaces are equal to two or more times the diameter of the individual fibres. They are not very different in appearance from the fibres found in the tectorial membrane. They are to be contrasted with their more fibrous and dense appearance in fixed and stained preparations.

*Mesothelial Cells:* The mesothelial cells (1,000×) on the scala tympanic surface of the basilar membrane are shown in Fig. 15. They are spindle-shaped cells that course in a direction at right angles to the fibres of the basilar membrane. They form a fairly compact layer and each cell resembles a modified endothelial cell.

*Peripheral Nerve Fibres and Nerve Endings:* Nerve fibres (500×) are seen in a part of their course through the tunnel area in Fig. 16, as the external spiral bundle in relation to external hair cells. Fibres to the internal hair cells appear to the left in another focal plane, so detail of their course is unsatisfactory. A number of nerve endings of external hair cells are noticeable. Examination of the base of hair cells shown in Figs. 2, 3 and 4 reveals nerve endings usually for which the connection to the peripheral nerve has been severed. Fig. 17 (2,000×) serves further to illustrate the course of the nerves and their endings on external hair cells. In Fig. 18 (1,000×) two different types of nerve endings are

shown in relation to internal hair cells. In one instance (to the right) there is a fine bifurcation of fibres, while to the left of this is a knob-like ending that appears to be single in this instance. Actually these endings are in clusters of two or more. They do not end on the base of the cell but pass somewhat higher and onto the side of the cell and end between cells. Their probable significance will be discussed later.

*Tectorial Membrane:* Usually the tectorial membrane was studied in preparations for which pieces of it had been isolated and mounted. A view of the appearance of such a strip under low power magnification (108×) is shown in Fig. 19. The border to the right was in contact with the limbus, while that to the left was in contact with Hensen's cells. At various levels of focus and with higher power it was possible to visualize certain features, such as the stripe of Hensen, the fibres that comprise the membrane and the network on its surface (see Fig. 20, 1,000×). In this photomicrograph only a small area of the net on its surface and the peripheral border are shown. The next extended over the peripheral half of the membrane, and its strands diminished in size at a point over the hair cells. The substance of which the net-like strands was comprised appeared to be similar to that seen on the border of the membrane overlying Hensen's cells.

## DISCUSSION.

In our opinion the results presented justify the use of the phase contrast microscope in the study of the organ of Corti. It is planned to extend the studies to include other parts of the inner ear and in addition to use other methods, such as electron microscopy. There is no organ in the body for which more observations have been erroneously described as pathologic changes than for the internal ear. Engstrom[6] has recently emphasized the need for a revision of older observations on the labyrinth by the use of newer methods. The need for detailed information regarding finer cell structure is further stressed by the progress now being made in the understanding of the physiologic principles of hearing (Davis and Tasaki[7]).

In the freshly isolated preparations of the organ of Corti there was no tendency for the hair cells to swell or change their shapes. The elongated and narrow external hair cell showed a tendency to bend in the middle third (portion above nucleus), but no distortion of it such as described for certain theories of its stimulation was discernible. The rather firm anchorage of the upper ends of the internal and external hair cells in the reticular membrane and the obviously rigid support of the phalangeal cells and pillar cells tend to produce a unit that is compact and not readily broken up. The anchorage of the organ of Corti to the basilar membrane is not an impressive feature, because it is readily lifted from the latter, as also occurs under experimental conditions, such as acoustic trauma. That the hair cells are the most fragile components of the organ of Corti and more susceptible than other cells to trauma and various agents such as drugs, toxins and so forth, is further borne out by attempts to tease and isolate single hair cells in fresh preparations. The body of the hair cells was usually torn away from the reticular membrane, leaving its cuticular border with the hairs intact. The remainder of the cell thus became lost, probably because it disintegrated rapidly. The preparations that had remained under observation for about one hour began to show progressive stages in deterioration, first in the hair cells and later in the supporting cells. No intracellular fibrillar network was in evidence in freshly mounted preparations. The hair cells appeared quite clear with granules, mitochondria and a ground substance of a nonrefractile type. A semisolid phase in which the ground substance and granules participated became conspicuous with age and deterioration of the preparation. It is possible that fibrils may be present but remain undetected by phase microscopy.

Hensen's cells are regarded as important in their function as supporting cells. Several features are presented in these observations to throw some doubt upon their value as supporting elements for the organ of Corti. In dissection of the organ of Corti they are readily removed in a strip showing that they have no firm anchorage to the reticular membrane

or basilar membrane. Perhaps a more plausible explanation of their position is not primarily that of a protective or supporting function but instead a physiological rôle in the maintenance of sensory and other supporting cells of the organ of Corti. Further work is at present under way to determine the nature and fate of the globules and granules revealed in these cells by phase contrast microscopy.

Hilding's[5] excellent description of the tectorial membrane cannot be improved upon by our observations. The net on the surface of the membrane observed by him and also De Vries[8] appears to be comprised of a homogenous substance and blends into the border of a similar material that is described by Hilding as overlying Hensen's cells.

The fibres of the tectorial membrane are similar in appearance to those observed by Katsuki, Mizuhira and Yoshino[9] using contrast microscopy on the cupula of the lateral line organ of fish. They are also similar to the fibres in the basilar membrane of the guinea pig with considerable interfibrillar substance apparent in each structure. In the process of preparing sections of the cochlea this interfibrillar material stains deeply for the basilar membrane and imparts to its appearance that of a dense fibrous structure. For the tectorial membrane the shrinkage and lack of staining of the interfibrillar material reveals a more delicate structure particularly subject to artefacts. The difference between the two structures is probably not in their fibrillar content so much as in their interfibrillar material.

The course of the peripheral nerve fibres in the organ of Corti was observed in many preparations. It was found that the nerve endings of the external hair cells are of one type, while the internal hair cells revealed two types of endings. The unusual ending was a cluster of small knob-like ramifications of the nerve fibres that did not attach itself to the base of the cell but instead was found between internal hair cells. The significance of this type of ending is not clear. Kolmer[10] described two types of endings in the maculae and cristae of human labyrinths but did not observe both types in the organ

of Corti. Katsuki and Yoshino[11] described two types in the cells of the lateral line organ of fish in preparations furnished them by H. Yamada. Perhaps these knob-like endings between the internal hair cells are the termination of the efferent fibres described by Fernandez[12] for the bundle of Rasmussen, but the explanation of their presence remains a problem for further study.

## SUMMARY.

The organ of Corti as studied under phase contrast microscopy has revealed information regarding its finer cell structure.

The hair cells are held in place by supporting cells and the reticular membrane. They are the most fragile components of the organ of Corti. They do not change their shapes readily even in preparations that show deterioration. They contain relatively clear ground substance of a nonrefractile type except for granules. Preparations that were about one hour old revealed some deterioration of the cell usually manifested as a semisolid phase of the cytoplasm.

The tectorial and basilar membranes are comprised of fibrils with intervening interfibrillar substance that is probably of different chemical composition in the two structures.

The supporting function of the cells of Hensen is questioned. Further studies are under way to determine the nature of the globules and granules that are seen in the cells with phase contrast microscopy.

Peripheral nerve fibres and their endings are described for the organ of Corti. Two types of endings were observed for the internal hair cells. Their significance is not clear.

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# THE ABSENCE OF RESIDUAL EFFECTS ATTRIBUTABLE TO THE OTOLITH ORGANS FOLLOWING UNILATERAL LABYRINTHECTOMY IN MAN.

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When, under proper circumstances, a healthy person is subjected to a change in direction of resultant force relative to himself, he experiences illusions of a striking nature. Although his position relative to the earth's axis remains the same, he feels as if it were undergoing a change, and objects stationary with respect to himself appear to move and to assume new positions in space. A remarkable feature of these illusions is that they are susceptible to quantitative measurement. Such measurements can be made in terms of the angle through which the apparent movement takes place, the obtained value can be compared not only with another experimental datum but also with the actual angle through which the resultant force moved. It is obvious that these illusions are the result of stimulation of sensory receptors which respond to a change in direction of resultant force and that they represent an attempt at reorientation in accord with the changing lines of force. The sensory organs which respond specifically to this stimulus are the otolith bodies, and it has been stated[15] that persons without otolith organs do not ex-

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Research Report. Project Number NM 001 059.01.33.

Approved by Capt. Ashton Graybiel, M.C., U.S.N., Director of Research.

Released by Capt. James L. Holland, M.C., U.S.N., Commanding Officer.

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perience these reorientation phenomena. Insofar as this is true, these illusions may be used as indicators of the functioning of the otolith organs.

Although these illusions were described many years ago by Purkinje[13] and Mach,[9] it is only recently that they have been the object of intensive study.[3,5,6,20] The apparent movement and displacement of objects in the visual field has been termed the oculogravic illusion.[5] Under favorable conditions there is good correspondence between the rate of apparent movement and the magnitude of apparent displacement in space. It behaves in an orderly manner and, in the case of sophisticated subjects, the test-retest reliability in estimating the magnitude of the displacement is good. The oculogravic illusion is not due to eye-movement; it may be related to the ability to perceive space.[19]

The apparent change in bodily position consists of a feeling of being reoriented in space. The reorientation also applies to the various supporting structures such as chair and floor. Under favorable experimental conditions, the change occurs slowly but is very clearly perceived.

In the experiments now to be reported, the responses to a change in direction of resultant force were compared in two groups of subjects under conditions favorable for the estimation of these two illusory effects. The subjects in the patient group had previously undergone unilateral labyrinthectomy; the subjects in the control group were healthy.

## SUBJECTS AND PROCEDURE.

Ten female subjects participated in the experiment. Five were healthy and had never suffered from any disease or disorder involving the inner ear. In the remaining five, unilateral labyrinthectomy had been performed in the course of treatment for Ménière's syndrome; four right and one left. Three of the subjects had no complaints; one still experienced rare mild attacks of vertigo and one suffered frequent attacks. The clinical findings in these five cases have been described elsewhere in detail.[8]

The experiments were carried out on a human centrifuge of the inertia wheel type.[3] The velocity of rotation of the centrifuge platform was graphically indicated by means of an Esterline Angus Recorder (E-A). A modified airplane gunner's seat was mounted on the centrifuge platform, 15.86 feet away from the center of rotation. The seat could be rotated around a vertical axis and secured in any of four positions in relation to the center of centrifuge, namely, *1.* facing center, *2.* facing forward (90° to the right of center in the direction of rotation of the centrifuge), *3.* facing away from the center (outboard), and *4.* facing backward (90° to the left of center and opposite to the direction of rotation). An interphone allowed two-way communication between subject and operator.

Attached to the seat was a metal frame on which were mounted a target light, biting board, and hand grips. The target light was a modified collimated star.[2] The visual target consisted of two lines of light in the same axis but separated by a short distance. The target could be rotated around an imaginary point dividing the space by which the lines were separated. Clockwise or counter-clockwise rotation was effected by means of a reversible motor under the control of either subject or experimenter. The target was geared to a selsyn transmitter which activated a large needle on a dial calibrated in degrees and situated in the control room. Two pistol-grip hand holds with thumb-operated push-button switches were conveniently placed for the subject's use. Depression of the push-button on the right caused the target to rotate clockwise and on the left, counter-clockwise.

### ESTIMATION OF APPARENT CHANGE IN POSITION OF A VISUAL OBJECT DURING EXPOSURE TO CENTRIPETAL FORCE.

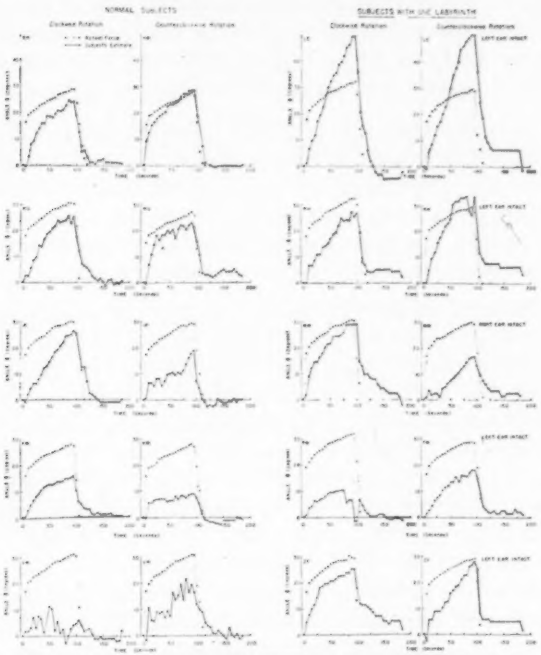
In these experiments the subject's task consisted of maintaining the lines of collimated light in what appeared to be the horizontal position during and for a short time after exposure to centripetal force (maximum angle $\phi \cong$ 30 degrees) for one and one-half minutes. The subject sat erect with head held firmly by means of the biting board and made the necessary adjustments by means of push-button

switches incorporated in the hand grip. The phenomenal horizontal was determined both before and after each experimental trial by offsetting the star and requiring the subject, who was shielded from all extraneous light, to return it to what she considered to be the horizontal position. In each instance the average of six offsets, alternately clockwise and counter-clockwise, was determined. Each subject participated in two trials facing forward with the star appearing to rotate clockwise and two trials facing backward with the star appearing to rotate counter-clockwise.

In each experimental trial, the readings on the dial were recorded every five seconds both during the period of acceleration and for a period of one and one-half minutes thereafter. The centripetal force was determined at the same five-second intervals and the angle $\phi$ calculated for comparison with the angle through which the subject rotated the star.

The results are summarized in the form of graphs in Fig. I. Each graph represents the mean values obtained on two trials. The curves have been "normalized" by using as the baseline the subject's phenomenal horizon rather than the true horizontal position. This procedure was equivalent to a translation of the entire curve up or down the ordinate by an amount equal to the subject's error in estimating the horizontal position of the line. A typical "good response" was made by the normal subject BW when observing counter-clockwise rotation. This indicates that she used her original position as the reference criterion for the horizontal and that she estimated the apparent change accurately. A typical "poor response" was made by the normal subject VW when observing clockwise rotation. It is altogether probable that she used her changing apparent position throughout the experimental trial as the criterion reference for the horizontal and set the target line to conform to it. The remaining responses represent greater or lesser degrees of compromise between these two extremes. That so many poor responses were made by the normal subjects is to be explained by their lack of training and experience in this procedure.[11] Almost equally "poor responses" were made by subjects in the control and patient

groups. The group differences are small and become insignificant in the light of the great individual variation shown in both groups. The evidence is insufficient that the subjects with one labyrinth responded differently when the application of force was from the right or left side.



APPARENT CHANGE IN DIRECTION OF HORIZONTAL LINE COMPARED WITH CHANGE IN DIRECTION OF THE RESULTANT FORCE (ANGLE $\phi$). EACH POINT IS THE AVERAGE OF TWO OBSERVATIONS

FIGURE I

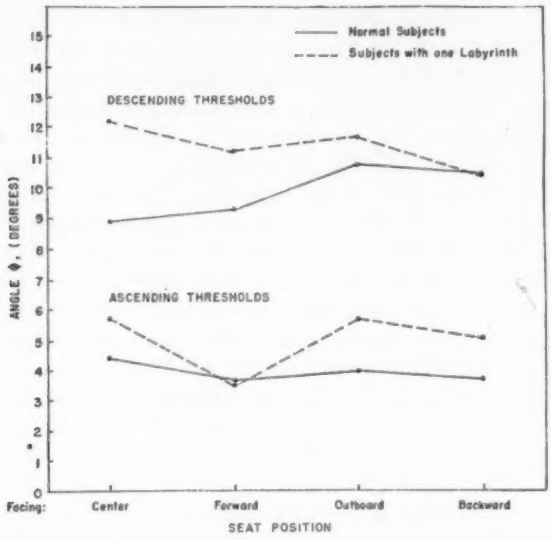
A comparison of the initial and final target settings shows a better correspondence in the case of the control than in the patient group. Two subjects in the patient group are responsible for nearly all of the difference. The significance of this finding is in doubt because of the small number of subjects tested.

ESTIMATION OF APPARENT CHANGE IN BODILY POSITION DURING EXPOSURE TO CENTRIPETAL FORCE.

*Threshold Measurements.* In this series of experiments the subject, with her eyes closed and in the dark, was required to signal the moment she first perceived that she was tilted away from the vertical and the moment that she returned. The velocity of rotation of the centrifuge was slowly increased from zero to 7 r.p.m. using a constant throttle setting, after which the engine was disengaged, by means of the clutch, which caused the velocity of rotation to decrease slowly by virtue of the friction in the bearings. The same pattern of acceleration and deceleration was maintained insofar as possible in all experiments. During the first series of experiments the subject sat erect with head held firmly by means of the biting board. The subject was requested to close her eyes and a closely-fitting goggle with opaque lenses provided further protection against external cues. The subject, although not always aware of the onset of rotation, soon felt she was tilting or leaning away from the vertical. The direction of tilt depended upon her position on the centrifuge. The subject on perceiving the tilt signalled the operator, who recorded the velocity at that moment by means of a signal recording on the E-A. During deceleration the subject signalled again when she felt that she was upright and in her original position. Each person was subjected to five experimental trials in all four positions. The initial seat position was randomized insofar as possible, and the five experimental trials were given as a continuous series. Between trials the subject was allowed a two-minute period of relaxation.

The results are summarized in Fig. II. It is apparent that there are no significant differences between the two groups. The similarity in the case of the ascending thresholds, which are more meaningful than the descending thresholds, is striking. The values for the subjects in both groups are slightly lower than in another experiment where highly sophisticated subjects were used.[3,6] In the case of the descending thresholds three of the normal subjects had slightly lower values

than the rest. The group differences, however, are small and unimportant. The maximum values recorded were similar for all subjects in all positions.




MEAN ANGLES (Φ) AT WHICH TILT (ASCENDING) AND RETURN TO VERTICAL (DESCENDING) WERE FIRST PERCEIVED IN VARIOUS SEATING POSITIONS. EACH POINT IS THE AVERAGE OF FIVE OBSERVATIONS.

FIGURE II

*Suprathreshold Determinations.* In carrying out these tests the procedure was altered in some respects. Two machinist's combination squares were mounted on the framework of the seat within easy reach of the subject's hands. The "square" consisted of a one-foot grooved metal rule which could be rotated in relation to a protractor head which was scored to read in degrees. In all experimental trials the protractor was secured at the middle of the rule. One square was placed with the rule horizontal and the other with the rule vertical.

In both cases the subject was placed so that she could grasp the ends of the rule and rotate it about its midpoint. Determination of the true horizontal and vertical positions was carried out by means of a level and plumb bob, respectively. Each subject was tested under static conditions and was required, with eyes closed, to adjust the bar within a period of 15 seconds. After a short period of indoctrination, 10 adjustments were made with each bar during the static condition. Following this, the subject was suddenly exposed to a centripetal force (angle $\phi \cong 30$ degrees) for one minute and 30 seconds. She was then required within 15 seconds to adjust the horizontal bar to what she considered to be the horizontal; 10 seconds later, she adjusted the vertical bar to what she considered was the vertical. Each subject made these adjustments during five consecutive experimental trials in each of the four different seat positions. The subject had a brief period of relaxation between trials. Seat position order was randomized.

The results are summarized in Table I. A distinction has been made between the primary and secondary adjustment in each experimental trial. For example, if the direction of centripetal force was such that the subject felt as if she were tilting backward, the primary adjustment would be made with the vertical rod which moved in the same plane as the lines of force, and the secondary adjustment would be made with the horizontal rod which moved in a plane at right angles to it.

If the results are evaluated in terms of the accuracy with which the subjects indicated the change in direction of resulting force, it is obvious that none performed well in all four positions. Some made an accurate adjustment in certain seat positions but not in others. Under-correction was a more frequent response than over-correction, but sometimes no correction was made. One subject (PW) did not make any primary correction for all four seat positions. This subject belonged to the abnormal group and her failure to respond deserves special comment because it could be interpreted as indicating nonfunction of the remaining otolith organ. The

TABLE I.

AVERAGE ERROR OF SUBJECTIVE ESTIMATES OF THE VERTICAL AND HORIZONTAL PARALLEL WITH (PRIMARY ADJUSTMENT) AND PERPENDICULAR TO (SECONDARY ADJUSTMENT) THE PLANE OF RESULTANT FORCE.

| | PRIMARY ADJUSTMENT | | | | SECONDARY ADJUSTMENT | | | |
|---|---|---|---|---|---|---|---|---|
| | Vertical | | Horizontal | | Vertical | | Horizontal | |
| | Facing Center | Facing Outboard | Facing Forward | Facing Backward | Facing Forward | Facing Backward | Facing Center | Facing Outboard |
| NORMAL | | | | | | | | |
| B. W. | 22.4 | —21.0 | —26.2 | +25.2 | 4.8 | 6.0 | 1.2 | 2.5 |
| M. G. | 18.7 | —18.1 | —27.9 | 26.7 | 6.3 | —0.1 | —1.7 | —0.6 |
| I. P. | 24.3 | —19.2 | —18.3 | 18.1 | —0.8 | —2.5 | 1.0 | 0.8 |
| K. M. | 22.8 | —24.9 | —30.1 | 29.3 | 7.7 | —0.2 | —0.2 | 0.0 |
| V. W. | 4.7 | —23.7 | —19.2 | 23.9 | 8.0 | 6.2 | —0.5 | 0.2 |
| OPERATED | | | | | | | | |
| L. E. | 24.1 | —25.4 | —26.3 | +31.7 | 6.5 | 1.8 | 2.2 | —1.6 |
| R. H. | 25.8 | —26.9 | —27.7 | 24.9 | —3.1 | —6.8 | 2.6 | —1.2 |
| M. W. | 14.9 | —22.0 | —19.3 | 17.8 | 2.5 | 5.0 | —0.1 | —0.7 |
| P. W. | 29.3 | —30.7 | —27.2 | 35.3 | 0.1 | —6.2 | —0.5 | —1.0 |
| Z. F. | 24.4 | —22.1 | —25.3 | +33.6 | 6.5 | 5.0 | —0.3 | —1.2 |

NOTE: Plus indicates clockwise rotation of test rod; minus indicates counter-clockwise rotation of test rod.

chief argument against this conclusion is the fact that she made two fairly good adjustments when the visual target was used. A further argument is the fact that the consistency of her response was good. The difference in responses between the two groups is insignificant.

## DISCUSSION.

Unilateral labyrinthectomy or its equivalent has been frequently exploited in attempts to unravel the rôle of the non-acoustic portion of the labyrinth. In analyzing the results of such experiments the following first-order variables must be kept in mind: *1.* two organs of special senses are involved, namely, the semicircular canals and the otolith bodies, the separate functions of which can be studied best by selective stimulation or selective suppression, *2.* the immediate effects following operation differ from the residual effects, *3.* the results differ according to the animal species.

Both in animals and in man the immediate results of unilateral labyrinthectomy are severe. There is good evidence that, in certain animals at least, the symptoms arise in part on the injured side and in part from unbalanced effects from the remaining labyrinth.

The residual effects of unilateral extirpation vary greatly depending upon the animal species. In general, the higher the level of phylogenetic development the smaller the residual effects.[10] Thus the frog compensates poorly, the pigeon moderately well, and the dog very well. Compensation involves different mechanisms, but the cerebral cortex plays the dominant rôle.[1]

Of particular interest to us here are the residual effects which might be ascribed to the otolith organs. In animals some of the most characteristic effects following unilateral labyrinthectomy are the forced position of the head, trunk, eyes, and sometimes other portions of the body. These effects decrease gradually following operation and in some animals disappear entirely, but in others a permanent residuum is left. That these effects are of otolithic origin has been shown

beyond doubt.[16] They are considered to be due to the uncompensated reflexes arising in the remaining otolithic organ.

It is clearly evident that the results of the experiments on animals cannot be applied directly to man; although in terms of man's position in the phylogenetic series, it would be expected that compensation following unilateral labyrinthectomy would be extremely good. Evidence of residual effects might be sought under static or dynamic conditions. Elsewhere[12] we have reported the results of a static experiment wherein the postural mechanisms were placed under stress. The same subjects were used as in the present experiment, and no differences in response were observed regardless of whether the principal stress fell on the operated or normal side.

In dynamic tests the otolith organs may be stimulated by tilting the subject's head in relation to the lines of gravity or changing the direction of the lines of force with the subject's head remaining fixed. Gollas[4] studied seven patients by measuring the counter-rotation of the eyes. When the patients were inclined toward the side of the nonfunctioning labyrinth, the counter-rotation of the eyes was normal, and when they were inclined toward the normal labyrinth, the counter-rotation of the eyes was diminished. From this it was concluded that although the remaining labyrinth acts upon both eyes, the effect is unequal. Spiegel and Sommer,[14] utilizing Grahe's table, also carried out experiments on subjects with a single functioning labyrinth. Following a tilt toward the operated or normal side, subjects were required to estimate the vertical position. The results indicated a displacement of the phenomenal vertical toward the injured side. Thetford and Guedry[17,18] were unable to confirm these findings in our subjects. Utilizing a tilt room, they found no displacement of the phenomenal vertical toward the injured side and no significant differences in response between normal subjects and those with a single labyrinth.

Dynamic experiments similar to those described in this report have been carried out using deafmutes as subjects.[1] When exposed to a change in direction of the lines of force,

many of them did not give any indication that they reorientated themselves in accord with this change. It was concluded that failure to do so was attributable to the absence or nonfunctioning of the otolith organs. If this conclusion is accepted, it furnishes a strong argument that such a procedure is adequate to test the function of the otolith organs. Our negative results, therefore, are significant and allow the conclusion that under the conditions encountered in our experiments, persons with a single otolithic organ respond the same as normal persons with a synergic pair. Stated differently, the evidence suggests that compensation following the loss of the otolith organ on one side is, for practical purposes, complete.

## SUMMARY.

Healthy subjects and subjects who had previously undergone unilateral labyrinthectomy for Ménière's disease were exposed to centripetal force on a human centrifuge. The change in direction of the lines of force relative to the subject produced striking illusions wherein objects in the visual field appeared to change their position in space, and the bodily position seemed to have changed. A comparison of the responses made by the subjects in the two groups revealed insignificant differences. It was concluded that under the experimental conditions defined, subjects with a single labyrinth respond normally when gravity receptors are stimulated. If it is assumed that the otolith bodies are specifically involved, it may be further concluded that the residual effects attributable to the unilateral loss of these sensory organs are insignificant or nil.

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We wish to thank Miss Frances Clopton, Clayton S. Ezell, H.M.C., U.S.N., and the staff of the Acceleration Unit of the U. S. Naval School of Aviation Medicine for technical assistance. We are also grateful to Theo. E. Walsh, M.D., St. Louis, Mo., who arranged for the cooperation of the subjects in this study.

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# THE USE OF DIHYDROGENATED ERGOT ALKALOIDS IN OTOLARYNGOLOGY: A PRELIMINARY REPORT.*

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A consideration of the pathologic physiology involved in certain otolaryngologic disorders would indicate that blockage of the sympathetic nerve supply to the area involved might be of therapeutic value. Our purpose is to present a preliminary report on the usefulness of such a sympathetic inhibitor — a combination of dihydrogenated ergot alkaloids known as Hydergine(R). The number of cases of each type we have to report (see Table I) is admittedly small, but as far as we know, this is the first report to appear in the English literature on the use of this drug in otolaryngology. We hope this factual report will stimulate a more widespread evaluation of this medication and of the therapeutic approach used.

TABLE I — TYPES OF CASES.

| | |
|---|---|
| Vertigo | 37 |
| Hypertensive epistaxis | 11 |
| Dryness of the mouth | 13 |
| Glossodynia | 11 |
| Postnasal discharge | 17 |
| Atrophic rhinitis and nasopharyngitis | 12 |
| Miscellaneous | 15 |
| Total | 116 |

*From the Department of Otolaryngology, the Tulane University of Louisiana School of Medicine, and the Eye, Ear, Nose and Throat Hospital, New Orleans, La.

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## PHARMACOLOGY.

Ergotoxine, one of the alkaloids of ergot, has been shown to be composed of ergocornine, ergocristine and ergokryptine. In the natural state, their vasoconstrictor action overshadows their inhibitory effect on the sympathetic nervous system. Hydrogenation, however, causes the sympathetic blocking action to become predominant, the vasospastic action being virtually eliminated.[1] In addition, probably because of the relatively purer state of these compounds, toxic reactions from their use are almost nil.

In this clinical study, the preparation used was an equiproportional mixture of the three hydrogenated alkaloids of ergotoxine.* For the most part, the medication used was in the form of sublingual tablets, although, in some cases, parenteral administration and sublingual drops were resorted to. Each sublingual tablet contained 0.167 mgm. of each of the dihydrogenated alkaloids; each 1 cc. ampoule of the parenteral form and sublingual drops contained a like amount.

Among the pertinent pharmacodynamic actions of the ergotoxine group which have been reported by various investigators are:

*1.* Depression of the central vasomotor centers with resultant vasodilatation and, in some cases, a fall in blood pressure.[2]

*2.* Peripheral vasodilatation.[3]

*3.* Central inhibition of pressor-receptor reflexes which bring about compensatory vasoconstriction and arterial blood pressure rise.[4]

*4.* Centrally induced bradycardia; this is rather unusual among sympathetic blocking agents.[5]

*5.* Central sedative action; also augmentation of barbiturate action.[6]

*6.* An adrenosympathicolytic effect.[7]

*Provided for this study by the Sandoz Chemical Co. Hereinafter referred to as Hydergine for the sake of brevity.

*Toxic Manifestations:* In the course of this study, only four persons reported untoward manifestations which were attributed to the drug. Three of these suffered with nausea which was not severe enough to warrant withdrawal of the medication. The fourth experienced weakness, slowing of the heart rate, nausea and headache; in this case the drug was discontinued.

## VERTIGO.

Hilger and Goltz[8] and Williams[9] have very brilliantly described what might be called "the vasomotor syndrome of the inner ear."

Hydergine was administered to 37 patients whose vertigo was ascribed to a vasomotor imbalance of the inner ear. Following a thorough history, a complete ear, nose, and throat examination, caloric tests, and, in some instances, audiograms were done. In 33 instances there was marked to complete remission of symptoms; four patients reported little or no improvement. Williams urges the use of a combination of therapeutic agents in such cases; in the vast majority of our cases, no medication other than Hydergine was used.

The following are typical case histories:

Mrs. O. H., age 70. This patient gave a history of having frequent attacks of postural vertigo for six weeks prior to her first visit. No history of hearing loss or tinnitus. Physical examination of the ears, nose and throat was negative. The Eustachian tubes were freely patent bilaterally. Caloric tests revealed hyperactivity of the left labyrinth. The patient stated that the vertigo caused by the caloric test, while similar to that spontaneously experienced, was not quite so severe. She was given one sublingual tablet four times a day. At the end of one week, she reported 90 per cent improvement. During the second week of therapy, she experienced only two mild attacks; since then, she has been asymptomatic.

Mrs. L. K., age 29. This patient experienced a sudden onset of recurrent attacks of postural vertigo and nystagmus. At about the time of onset of this condition, there was angioneurotic edema of the upper lip on the left. An audiogram revealed mild depression of the threshold for low tones by air conduction. Hydergine sublingual tablets were started, and within 48 hours the nystagmus had ceased and there was a marked reduction in the amount of vertigo. At the end of 96 hours the patient was asymptomatic. Following a week of therapy, the medication was stopped by the patient and she promptly became vertiginous again within 24 hours. The medication was resumed and control was again achieved.

F. G., age 53. For two months prior to the first visit this steel construction worker had been experiencing repeated episodes of vertigo of

sudden onset, diminished hearing and tinnitus. There was a past history of multiple allergic manifestations and his nasal smear revealed a two-plus neutrophilia and one-plus eosinophilia. Physical examination of the ears was negative, the Eustachian tubes were patent bilaterally, the nasal mucosa was pale and the nasal septum was markedly deviated, and the tonsils were chronically inflamed. Audiometry revealed a typical "boilermaker" type of hearing curve bilaterally, consistent with his having worked in a noisy environment all of his life. A caloric test revealed hyperactivity of the left labyrinth, the vertigo produced being identical with that occurring spontaneously. Hydergine sublingual tablets were given, and the patient reported marked improvement within one week, although his postnasal discharge was somewhat increased.

*Comment:* Because the inner ear is enclosed in an almost rigid bony box, its function may be disturbed by even such slight pressure changes as are brought about by alteration of blood vessel tone. The significance of vascular disturbances is better appreciated when one recalls that the blood supply of the inner ear is derived from end-arteries. Depending on the vessel, or vessels, involved, the patient may experience vertigo, hearing loss, or tinnitus, or a combination of these symptoms.

The most widely-held present-day concept of the pathologic physiology in these cases is that there is arteriolar spasm with dilatation of the capillary-venule network distal to the spastic segment. In the distended loop, it is felt that there is sludging of blood, liberation of toxic metabolites, among which is histamine, anoxia of the blood vessel walls, and increased capillary permeability which leads to tissue edema and possibly increased accumulation of endolymph.

The best treatment is, of course, removal of the causative factor. To restore homeostasis quickly, however, an attack should be made on all aspects of the problem. Thus, a vasodilator would seem indicated for relief of vascular spasm, an antihistaminic drug to combat the effects of histamine, decreased salt intake and a salt-free diet to help reverse the edema, sedation to control the psychosomatic element, an anticholinergic agent for the parasympathetic preponderance, attempts at acidification to combat the effects of alkalinity on the autonomic nervous system, and the administration of vitamin C for its effect in reducing the permeability of the

capillary walls. For this reason, we feel Hydergine was given a particularly rigid test in the vast majority of instances, inasmuch as no other medication was used.

One may theorize that the effectiveness of Hydergine in these cases is the result of a combination of its pharmacodynamic actions. The vascular spasm is probably relieved by the peripheral vasodilatation. Once relieved, the spastic state is probably prevented from recurring by depression of the central vasomotor center. Further, the central sedative effect undoubtedly serves to allay some of the psychosomatic element usually present in these conditions.

Three other actions of Hydergine may be of some importance. Fries *et al.*,[3] while studying limb blood flow, have reported that during therapy there is usually a significant decrease in vascular distensibility, increase in venous pressure, and increasing prominence of arterial pulsations in antecubital venous recordings. It is possible that these changes may promote resorption of transudate resulting from vasomotor imbalance.

*Administration:* All patients received sublingual tablets every four hours.

### EPISTAXIS OF HYPERTENSIVE ORIGIN.

Severe epistaxis associated with hypertension and arteriosclerosis frequently proves to be a troublesome and difficult therapeutic problem. Such cases often continue to hemorrhage in spite of the routine nasal and postnasal packing, sedation, and the administration of the usual coagulating substances. It appears that bleeding does not stop until the blood pressure is lowered from loss of blood; therefore, more than merely stopping the bleeding is required; attempts to reduce the blood pressure would seem logical. It was for this latter purpose that we employed Hydergine in 11 cases. We feel that our results have been very good; we should like to emphasize, however, that this medication is not a substitute for sedation and packing, but its use appears warranted in conjunction with these time-proven methods. The following is a typical case history:

N. K., age 54. First seen at 10:15 P. M. on April 12, 1952, while hemorrhaging profusely from the left nostril. There was a long history of hypertension, for which he had been placed in retirement by the New Orleans Fire Department. Recently he had been experiencing considerable emotional upset, and, prior to the onset of bleeding, he had noted the return of such symptoms as headache, easy fatigue, tinnitus, and some dyspnea in spite of strict adherence to the regime prescribed by his internist. Much of his anxiety on admission could be traced to the fact that one year previously he had been hospitalized for three weeks, during which he had undergone several traumatic attempts at nasal packing and he feared a repetition of this.

On admission, there was profuse hemorrhage, both from the anterior and posterior nasal openings on the left. The bleeding point could not be identified. B. P. 224/120, pulse 110, respirations 25, and temperature 98°. Hemorrhage was so profuse that 3 gr. of Luminal(R) were given and the patient was taken directly to the operating room, where both nasal and postnasal packing was introduced. This controlled the bleeding to a large extent, but there was still some oozing around the packing.

Upon returning to his room, the following orders were given and treatment begun: sedation, 400,000 units of penicillin daily, codeine gr. j for cough or pain, and Hydergine 1 cc. immediately and to be repeated every six to 12 hours or when indicated by a rise in blood pressure.

Progress notes:

April 13, 1952, 1:00 A. M.; B. P. 190/100. Small amount of blood oozing from around packing.

4:00 A. M.: B. P. 184/96. Resting well.

8:00 A. M.: B. P. 150/84. No bleeding. 1 cc. Hydergine intramuscularly.

11:00 A. M.: B. P. 146/84. No complaints.

2:00 P. M.: B. P. 134/78. Pulse 90. Respirations 20. Nourishments taken well.

6:00 P. M.: B. P. 144/70.

8:00 P. M.: B. P. 148/72. Postnasal packing removed.

April 14, 1952, 8:00 A. M.: B. P. 138/84. A quiet night; no bleeding. Nasal packing removed. 1 cc. Hydergine intramuscularly.

1:00 P. M.: B. P 165/90. 1 cc. Hydergine intramuscularly.

4:00 P. M.: B. P. 150/84. 1 cc. Hydergine intramuscularly. Sublingual Hydergine begun.

8:00 P. M.: B. P. 140/84. No complaints.

April 15, 1952: A good night; no bleeding. Discharged with orders to continue the sublingual Hydergine tablets until again seen by his internist.

*Comment:* As Schroeder[10] has pointed out, most cases of hypertension are now felt to be of psychosomatic origin; those which are not undoubtedly have developed some psy-

chosomatic overlay. The psychosomatic disturbance is said to be mediated through the autonomic nervous system in the form of relative sympathetic preponderance with resultant generalized vasoconstriction of the vascular bed. In the kidneys, vasoconstriction causes elaboration of long-acting pressor substances into the circulation. Long-sustained hypertension will lead to pathologic change in the arterial walls; this is of especial importance in the case of the kidneys, as organic renal ischemia may ensue.

If this theory is correct, a rational approach to the problem would consist of blockage of the neurogenic sympathetic influence and inactivation of the pressor substances in the blood.

Because the emotional element looms so large, both as a precipitating and perpetuating factor, in the average case of hypertensive epistaxis, we believe that control of the neurogenic sympathetic influences in these crises should receive the highest priority.

The use of Hydergine in these cases is predicated on its ability to depress the sympathetic neurogenic influences, to provide peripheral vasodilatation, to provide central sedation (in addition to augmenting barbiturate action), and to lower the blood pressure; all these effects are obtained with the production of none of the signs of vasomotor collapse.

In regard to the blood pressure, marked lowering should not always be expected, nor does it always appear necessary for good results and alleviations of symptoms in hypertension. When there are organic vasculorenal changes, the drop in pressure will not be so great; generally speaking, it will be proportional to the extra load occasioned by the psychosomatic overlay. The important factor in this aspect of the treatment of hypertensive epistaxis seems to be to lower the blood pressure to a level which the blood vessels are able to withstand without a break occurring; apparently healing of the broken vessel cannot occur until the pressure falls below this critical level.

Most patients claim subjective improvement; objectively, they seem in better spirits, display an improved attitude towards their illness, and have a better mental outlook.

*Administration:* Following sedation and the necessary nasal and postnasal packing, 1 cc. of Hydergine is administered intramuscularly and the blood pressure recorded hourly. The dosage is repeated every six to 12 hours, depending upon the clinical progress of the patient, until the nose has been free of packing for 24 to 48 hours. Administration of sublingual tablets every four hours is then started and continued until it is certain that healing is complete; mild sedation may be used in combination with the sublingual tablets. The patient is then referred to an internist for prolonged medical management.

### DRYNESS OF THE MOUTH.

We have administered Hydergine to 13 persons whose complaint was dryness of the mouth. Twelve patients obtained complete relief of symptoms on Hydergine alone. The other case was partially relieved by Hydergine alone, complete relief being achieved when Hydergine was taken in combination with prostigmine bromide.

F. R., age 52. This patient was a visibly hypertonic type of individual who had been suffering with a sensation of dryness in the mouth for two weeks; this was accompanied by a decreased viscosity of the sputum. Questioning revealed that he was a bookmaker and that the onset of symptoms coincided with a decision on the part of the local law-enforcement agency to make things "hot" for him. Examination of the mouth was negative except for the fact that the mucosal surfaces had a dry, glairy appearance, and the saliva did, indeed, appear very white and concentrated. He was placed on one Hydergine sublingual tablet every four hours and reported relief within 48 hours; this did not coincide with a lessening of tensions as a result of his life situation. Following a week of therapy, there was no recurrence until 10 months later when the same situation again became manifest. Relief was again obtained with Hydergine in short order.

*Comment:* The salivary glands have both a sympathetic and parasympathetic nerve supply. They differ from other effector organs, however, in that both elements of the autonomic nervous system are cholinergic.[11] A decrease in salivary output, therefore, could be due either to a hypofunctioning autonomic nervous system or to hypofunctioning acini of

the glands (possibly, in some instances, at least, on the basis of a decreased blood supply). The effectiveness of Hydergine may be due to its peripheral vasodilator action, or to the depression of the central vasomotor system. In the case not responding to Hydergine alone, prostigmine bromide was used for its action in destroying choline esterase and thus permitting freer transmission of cholinergic impulses.

### GLOSSODYNIA.

Eleven patients who complained of painful, burning tongue were given a therapeutic trial of sublingual Hydergine. No organic basis for their complaints could be found, and there were no changes in the topography of the tongue. Eight patients reported rapid relief of symptoms, two obtained partial relief, and one patient noted no improvement of symptoms.

*Comment:* This symptom is most frequently thought of as being a manifestation of a deficiency state, either of niacin, riboflavine, pyridoxine, or a combination of these. It may also be associated with iron-deficiency anemia or organic nerve disease. Our cases, however, did not present any of the usual signs of the above-mentioned conditions.

Hydergine was used on the theory, in some cases at least, that the symptoms might be the result of a circulatory disturbance and would, therefore, be benefited by vasodilatation, the increased blood flow leading to improved nutrition and a "flushing out" of accumulated toxic metabolites. It is interesting to note that some of the vitamins used in the past are potent vasodilators.

### POSTNASAL DISCHARGE.

Hydergine sublingual tablets were used in the treatment of 17 patients who complained of a thick, tenacious, mucoid postnasal discharge. Repeated cytologic examination of the material revealed a large amount of mucus, a few mononuclear cells, some squamous epithelium, but no eosinophiles.

All patients reported marked improvement as a result of treatment. Questioning revealed they still had some discharge, but it was not thick and did not prove bothersome.

*Comment:* Individuals complain of nonpurulent postnasal discharge either *1.* when the amount of mucus is profuse, *e.g.*, as in allergic rhinitis, or *2.* when the mucus is thick and does not move normally along the path from the nasopharynx, down past the pharynx to be swallowed. In the latter instance, the patient becomes conscious of the thickened mucus and begins to make strenuous efforts to remove it. Sometimes an actual fixation develops and efforts to obtain relief become persistent, and often violent, and actually lead to nausea and vomiting.

Viscosity of mucus depends to a large extent upon the vascular supply of the region producing the mucus and the state of the secretomotor system supplying the glands. Proetz[12] has shown that when mucus becomes too viscid it will roll over in masses. It is at this point that the patient becomes conscious of it and symptoms begin.

Our rationale in trying Hydergine in these cases was based upon the hope that blockage of the sympathetic nerve supply would leave the parasympathetic secretory system unopposed, and, secondly, that the vasodilatation produced would improve the blood supply and thus re-establish normal viscosity. Our object was not to "cure," or abolish, the postnasal discharge; rather, it was to re-establish the normal mucus pattern.

### ATROPHIC RHINITIS AND NASOPHARYNGITIS.

Among the conditions in which the value of Hydergine was investigated was atrophic rhinitis and nasopharyngitis. The medication was administered to 12 patients. Two early cases of atrophic rhinitis remain asymptomatic, and the nose appears normal on inspection as long as the medication is taken. Seven moderately severe to severe cases report varying degrees of improvement while taking the drug; three of these report better results when Hydergine and prostigmine bromide are taken simultaneously. The criteria for improvement consisted of a decrease in crusting, both as reported by the patient and as seen by the examiner, and a diminished sense of dryness and fetid odor in the nose. Some patients who had cleansed their nasal passages once or twice daily with saline

solution found that this was necessary only about once weekly. The three cases which did not respond to treatment were among the most far advanced.

*Comment:* There is general agreement that in ozena there is a decrease in the blood supply to the nasal mucosa. Whether this is primary, or secondary to other factors, is not known; nevertheless, there have been reports concerning the usefulness of vasodilating agents in this condition. Hydergine alone, or in combination with prostigmine bromide, seems to be a useful palliative measure.

### MISCELLANEOUS CONDITIONS.

We have administered Hydergine sublingually in three cases of Bell's palsy, in several cases of nonallergic vasomotor rhinitis, and in a number of cases of seasonal hay fever.

All three cases of spontaneous Bell's palsy cleared almost completely within two weeks. One occurred in an infant to whom the medication was administered in the form of sublingual drops. One case recurred after discontinuing therapy, but it cleared rapidly after therapy was resumed. None of these cases showed a rapid response such as is sometimes said to occur during intravenous histamine therapy. It is realized that the response to therapy in these cases is difficult to evaluate because the rate of spontaneous remission is so high.

There seemed to be a favorable response in some cases of vasomotor rhinitis, but, without exception, symptoms were aggravated in all cases of seasonal hay fever.

### DISCUSSION.

In evaluating our results, the erratic clinical behavior of many of these disorders has been taken into account. We have kept in mind the fact that spontaneous regression may be sudden and complete, as it was in any case in our series; however, since the results were uniformly good, we believe this simple and safe form of medication should be widely tried and evaluated in the above and other otolaryngologic conditions.

Although we have used Hydergine alone in most of the cases, in order to obtain a true indication of its effectiveness we should like to emphasize that we do not consider it a panacea, and we firmly believe that in many instances better results might be obtained by using a combination of therapeutic agents. Thus, in vertigo, one may feel it necessary to combine a salt-free diet, antihistaminics, or some other well-established therapeutic measure, with autonomic blockage.

Again, in atrophic rhinitis this therapy may be combined with the local use of an antibacterial agent, high potency vitamins, estrogens, etc.

Because fundamental physiological studies are lacking, we realize that our explanations for the effectiveness of this particular preparation of dihydrogenated ergot alkaloids may be invalid. It is hoped, however, that this presentation may encourage others to try this approach so that the part played by imbalance of the autonomic nervous system in these disorders can be properly catalogued.

## SUMMARY.

*1.* A report is made of the results of a clinical evaluation of the use of a new adrenergic blocking agent, Hydergine, in several otolaryngologic disorders. Included among these disorders are: vertigo, spontaneous hypertensive epistaxis, dryness of the mouth, glossodynia, postnasal discharge, atrophic rhinitis and nasopharyngitis, and Bell's palsy.

*2.* The pharmacology of the dihydrogenated ergot alkaloids is reviewed and their pertinent pharmacodynamic actions indicated.

*3.* The rationale for the use of Hydergine in each disorder is presented.

*4.* It is emphasized that autonomic blockade by use of drugs is not considered a panacea in the conditions considered; rather, it should be considered another useful addition to our therapeutic armamentarium.

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# THE SIGNIFICANCE OF APHASIA IN OTOLOGY.*

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Hearing and speech have a close relationship. Children born deaf will remain speechless. Loss of hearing in early childhood will interrupt the normal development of intelligible speech. Monotony of diction, lack of emotional expression, agrammatism and a "telegram style" of conversation are the most frequent signs of this infantile type of aphasia.

The great neurologist of the last century, Wernicke, stated that the great majority of cases of aphasia was primarily an acoustic problem. His concept of the mechanism occurring when sounds are transmitted via the auditory nerve to the brain centers and are transformed into proper meaning and understanding is still brilliant today, 85 years after publication. From this tenet he came to the conclusion that the center of word understanding must be near the auditory center (Heschl's convolutions) in the temporal lobe. Aphasia will occur, he stated, when those fibres of the auditory nerve having the function of transmitting auditory stimuli to the sensory or receptive speech center are impaired.

Modern psychology cannot accept this idea as a working basis for diagnosis and therapy, since the research on aphasia has encompassed such a wide area, including optic aphasia, tactile aphasia, body topagnosia, etc. Limiting Wernicke's theory to the pure acoustic aphasia, we otologists might be inclined to appreciate its value.

The clinical picture of aphasia has many varieties and modifications. These manifestations depend upon the topographical location, extension, etiology, and the stationary or

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progressive character of the original brain lesion. Pierre Marie contributed the important psychological approach to the problem of aphasia in his concept that type, clinical course, prognosis and therapy are considerably influenced by the type of personality, the amount of education, and the degree of emotional stability of the patient. Modern speech experts agree that the proper rehabilitation of an aphasic patient is successful only when both aspects of the therapeutic approach, *i.e.*, the anatomical and psychological, receive due consideration.

The otologist participates in the aphasic problems when he has the opportunity to be called into consultation concerning aphasic patients; hence it may be proper to single out those types of aphasia which may originate from an otological lesion.

*1. Motor aphasia* (Bastian), also called "expressive aphasia" in the newer textbooks (Weisenburg and McBride). This aphasia was described by Broca, in 1861, as "L'Aphemie" (Speechlessness). He found its anatomic pathological topography in the third convolution of the left frontal lobe. Its predominant clinical feature is the patient's incapacity of spontaneous speech or difficulty in repeating words, although he understands all that is said to him. He may pronounce a few words expressing old inherent memories, *e.g.*, his name and birthday; or he may start to sing verses recalled from early childhood. He is conscious of his disability; he tries to pronounce a word repeatedly, and becomes angry and even desperate when unable to overcome his difficulties. Larynx and tongue do not follow his volition. He shows dysarthria and the heavy, thick speech so often associated with hemiplegic sufferers. Hours of depression may be followed by days of euphoria. He may sing, dance and kiss everyone in his environment. His whole personality changes; it regresses into childhood or even primitivity. He may begin to learn speech again like a child — as a paraphasic naming an object incorrectly, but similar in sound — or he attempts to express emotions in sentences without using articles, adverbs or adjectives in a so-called "telegram style." This telegram style

of speech is one of the characteristic symptoms of predominantly expressive aphasia. The patient knows his handicap, omits all small words which are not necessary for proper understanding of his thoughts and desires. He discards all expressions of abstract concepts and uses the simplest language directed to concrete objects and facts. The peculiar euphoria observed in this type of patient is considered to be a protective psychological mechanism against "catastrophic" conditions, *e.g.*, despondence, thoughts of suicide, etc. (Kurt Goldstein).

A sample of a typical motor aphasia is the following case which was seen by the writer:

M. S., a 56-year-old white male, German immigrant, was admitted to the Albert Einstein Medical Center, Northern Division, on Sept 4, 1951. He was in good health until two days prior to admission, at which time he became dizzy. The next day he dropped a coffee cup and believed he had lost the power in his right arm. A day later (Sept. 5) he became very restless and was unable to speak.

In 1946, the patient was treated by the writer for a fetid empyema of the left antrum. After several antral lavages and extraction of several decayed teeth, the infection cleared up. In March, 1951, the patient came to my office complaining of loss of taste and smell of two weeks' duration. In addition, he complained of severe headache over the left eye. Irrigation of the left antrum revealed a few flakes of mucoid pus. Smell and taste returned after a week. Roentgen studies of the paranasal sinuses were suggested several times, but refused by the patient for financial reasons. The possibility of a neoplasm was considered.

*Physical Examination*, Sept. 4, 1951: Well developed male, in a mild state of apprehension, unable to speak, except a few words which cannot be understood. In a state of excitement he says, "Ach mein lieber Gott" (My dear God); he cannot read nor write, not even his own name.

*Neurologic examination* revealed: weakness of the right lower face, no deviation of tongue, normal pupils, hyperactive reflexes of right upper and lower extremities, suggestive Babinski, right, no Hoffmann. Sensitivity of right side apparently less than left.

*Hematology:* Hgb., 16.5; R.B.C., 6,060 million; W.B.C., 9,850; poly., 60 per cent; mono., 2 per cent; lymph., 32 per cent; fil., 97 per cent; nonfil, 3 per cent. Blood sugar, 132; B.U.N., 15.

*Roentgen Studies of Skull:* Marked density of the left antrum and anterior ethmoid cells, no fluid level, no evidence of bone destruction (see Fig. 1).

*Diagnostic Impression:* Motor aphasia due to a vascular thrombosis in Broca's area. The writer was consulted concerning the possibility of an etiological connection of the sinus lesion with the cerebral accident. An active therapy of the left antrum (puncture or biopsy) was not performed at this time because of the general condition We had, however, the im-

pression that the Roentgen findings of the sinuses were the sequelae of the previous infection for which he was treated years before. Any connection of the motor aphasia with the antral lesion was doubted. Observation, however, was advised.

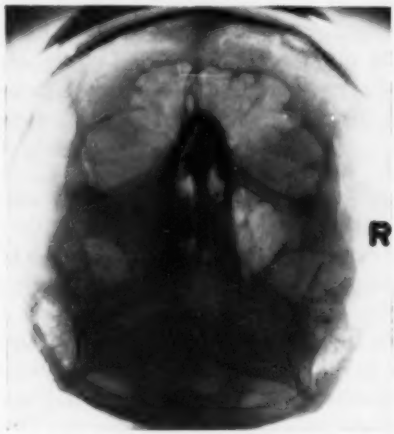


Fig. 1. M. H. Complete cloudiness of left maxillary sinus. There was no evidence of an etiological relationship between the motor aphasia and the antral lesion.

*Clinical Course:* The patient received three block anesthesias of the left stellate ganglion and was discharged on Sept. 20, 1951, to the family physician's further care. At this time he was able to sing old German songs, he could pronounce a few words (doctor, first names of members of family, etc.). He became euphoric for the next few days; then he said, "Go home, home."

Feb. 20, 1952, the patient came for a check-up. The sinuses were clear. There was no indication for local treatment. The patient has been attending the classes for speech therapy at Temple University Hospital since he was discharged from the hospital. He can speak again, but he still has difficulty pronouncing some words, especially nouns. He can write again, but is unable to play chess, at which he was once a master. He speaks in a telegram style, but with emotion. He follows a conversation without any difficulty and is hopeful of complete recovery.

Motor aphasia is rare in otological practice. Henschen found motor aphasia only twice in his 77 cases of temporal lobe abscesses. Haberer and Bonvicini described one case of temporal lobe abscess with destruction of Broca's area. They assumed an embolic etiology in this case. Körner and Gruber observed an aphasic with dominant motor features. Brunner

and Bénesi reported a mastoiditis complicated with a localized malacia of Broca's area. Brunner reported motor aphasia in a child which presented as an acute mastoiditis. Autopsy revealed a meningitis with encephalitic foci in the frontal lobe. A localized meningoencephalitis might have been the precipitating factor in other cases reported (Lucae, Heimann, Gorham, Lenz). Lorenz and Rudloff observed motor aphasia to disappear after drainage of an extradural abscess of the temporal lobe.

2. *Sensory aphasia* or recently coined "receptive aphasia" has also been listed in the literature under "Wernicke's aphasia," an entity caused by localized lesions of the temporal lobe. This author put the "center" of his sensory aphasia in the posterior portion of the first convolutions of the left temporal lobe. Henschen, the Danish neurologist, elaborated Wernicke's original idea. He dissected the cerebral cortex in mosaic-like fashion and established separate centers for the various functions of intellectual acts expressed in speech. Thus, he located the centers for amusia, agraphia, alexia, etc., and found that larger areas are involved than his teacher originally had assumed. Today Wernicke's aphasia is also called "temporoparietal aphasia," indicating particularly those areas encircling the posterior portions of the Sylvian fissure.

Wernicke's sensory aphasia is characterized by three clinical features: *a.* amnesia, *b.* agnosia and *c.* dysphagia. According to Poetzl the patient behaves as a normal person who enters a new country without any knowledge of the language. He hears sounds of words he cannot understand (acoustic agnosia); he does not know the names of objects shown to him (optic aphasia); he has forgotten names heard before (amnesic aphasia); he may not know how to read or write under the new conditions (agraphia, alexia); he has difficulty in pronouncing, spelling, grammar (paraphasia, dysphasia, dysarthria, agrammatism); he may persist in the wrong pronunciation despite the consistent effort of friends to help him overcome this handicap (perseverance). The only difference between the aphasic sufferer and the normal

newcomer is the fact that the sensory aphasic patient is usually not conscious of his deficiencies. That is also one main point in distinguishing the motor aphasic from this type.

The majority of Wernicke's aphasics suffered from vascular lesions. In most cases a partial thrombosis of the Sylvian artery was found. The posterior temporal branch supplying those speech areas is especially involved. Tumors and cysts may play an etiological rôle, but relatively seldom. The otologist sees the typical Wernicke's aphasia occasionally. A brain abscess will present all those signs mentioned if its extension has reached the more highly situated areas of the Sylvian fissure. This, however, is usually the case at the terminal stage only. Körner, in his relatively large group of 104 cases, saw the Wernicke type of aphasia only twice. If the abscess perforates deeper towards the insula (Island of Reil) and medially to the uncinate gyrus, an amnesic aphasia for taste and smell (gustatory and olfactory aphasia) can complicate the clinical picture (Henschen). Extension of the abscess towards the posterior portions of the temporal and anterior sections of the occipital lobe may bring a peculiar optic aphasia into the foreground. Such a patient does not remember the name of an object shown to him, but he will recall its name when he touches it (Zaufal-Pick, Siebenmann-Oppikofer, Singer, Manasse). Optic aphasia can also be combined with hemianopsia caused by a lesion involving the optic radiations (Cushing, Eagleton, Chavany). Optic and acoustic aphasia can be seen in the same patient (two cases in Oppenheim's series). Preysing's case, in which the patient could not recall the names of countries, towns and streets, is unique (topographic aphasia). Abscesses spreading anteriorly towards the frontal lobe may lead to a mixed type of expressive-perceptive aphasia. This has also been observed in many cases of vascular etiology.

*3. Global Aphasia* (Wepman). This designates a complete loss or disorder regarding speech understanding and expression. The consulting otologist often meets a difficult problem when confronted with the question: "Is this child's aphasia due to deafmutism or idiocy or other mental deficiency?" A

positive response to the caloric test and a positive cochleapalpebral reflex can prove that at least some vestiges of auditory function have remained. Its practical value, however, is limited because it is a qualitative test only. The modern psychogalvanometric test of Bordley and Hardy has now been accepted by the majority of audiologists as a great asset. This test is a conditioned reflex obtained by associating sound stimuli with electric shock. After conditioning is established, the sound heard will produce the skin changes which are normally elicited by electric shock. This response can be measured by the resistance of the skin to an electric galvanometer. In this way the threshold of sounds can be established without being dependent upon intentional response of the child; thus, it will help to establish the degree and type of deafness and many children will be saved from the fate of the global aphasic by obtaining the proper hearing aid and adequate speech-training.

A global aphasia in the adult can develop gradually. He may become deaf and later speechless. In former decades syphilis destroying large areas of the cerebral cortex was a contributor to this type of aphasia. In the otological literature few cases were reported with destructive lesions of both temporal lobes. The patients were deaf due to destruction of the hearing centers, and aphasic because of involvement of the speech intelligence centers (Poetzl, Mingazzini, Liepmann). Such a patient may present a "jargon aphasia," a type of language which probably has meaning for himself only (Jackson).

We must separate from this group those patients who became aphasic due to bilateral peripheral or central lesion deafness without involvement of Wernicke's speech centers. Liepmann called this aphasia "pseudoword deafness" to indicate the distinction from true "word deafness" or the typical acoustic aphasia of Wernicke. In the latter the acoustic apparatus still has preserved its physiological function to receive and transmit sounds. This is the case even if the left acoustic nerve is functionless by reason of the bilateral sound supply of the left acoustic center. Even in the case of destruction of

the left acoustic center, Wernicke's aphasia may not be the consequence, if the transcortical pathways connecting the normal right auditory center with the speech-center of the left hemispheres are intact.

*4. Amnesic Aphasia.* This aphasia appears in the literature under many designations: amnesia nominum (Henschen), amnesia verborum, anomia, amnesia verbalis acoustica, nominal aphasia, semantic aphasia (Head), acoustic agnosia, etc., This type of aphasia is seen most frequently by the otologist. According to Körner, it is one of the most important diagnostic signs of a left-sided temporal lobe abscess. Schmiegelow observed it 23 times in his 54 cases, Maier observed it 12 times in 14 cases. The pathological lesion involves the caudal portion of the left third convolution of the temporal lobe (E. Spiegel). Those areas lie opposite the petrous portion of the temporal bone, the sites of which are involved first in endocranial otogenous middle fossa infections. Hence, a brain abscess proper is not necessary to elicit amnesic aphasic disturbances. Görke, Grossmann and Laub observed them in cases of meningitis, assuming a mild encephalitis to be associated with it. Heine saw amnesic aphasia disappear after draining an otogenous subdural empyema of the middle cranial fossa. Brunner believes a retrograde thrombosis of the pial veins can suffice to elicit aphasic symptoms.

In the literature available to the writer he was able to find 26 observations of aphasia elicited by an extradural temporal lobe abscess: two had predominantly motor aphasia (Lorenz, Rudloff); five showed dysphasic symptoms (Salzer, Pritchard, Merkens, Jansen, Rudloff); two severe paraphasia (Heine, Alexander); and 12 various signs of perceptive aphasia (Alexander, Bönninghaus, Beck, Oppenheim, Goldflamm, Bach, Büch, Neumann, Altmann, Fremel, Frey, Lemaitre and Aubin). The other five cases presented a specific amnesic aphasia or nominal aphasia. Galtung saw an 11-year-old girl who had forgotten simple historical data and numbers she had just learned in school. Lorenz's patient presented an isolated amnesic aphasia; she could not name a few objects shown to her out of a large number of others. This specific anomia

persisted for some time. In Thormann's patient amnesic aphasia did not disappear until six weeks after exposure of the extradural abscess. Körner suspected an abscess of the temporal lobe. Several punctures and probatory incisions directed to various parts of the temporal lobe did not give evidence of an abscess. Henschen and Moulonquet found a general amnesic aphasia which disappeared shortly after the extradural abscess was opened. The writer had a similar experience. It may be justified to report the history, clinical course and diagnostic problems concerning this patient:

E. M., an 81-year-old white widow, was admitted on my service at the Albert Einstein Medical Center, Northern Division, on Aug. 18, 1950. She was conscious, could walk around and could stand upright. It was impossible to get a history from her. She complained of pain in the left ear and over the left side of the head (pointing to the left temple). She was entirely disoriented in time and place. Relatives and friends asking questions were answered with: I know you, but I don't know your name. She talked, but often she would begin a sentence without finishing it. The admitting resident stated, "Her sentences have no logical sense." On questioning, she would say, "I am three years old." A few minutes later she would answer, "I am 100 years old, maybe nearly 200." Objects (pencil, etc.) shown to her could not be designated by name. She was completely unemotional and showed no interest in things going on about her. She said, "I am not sick, but I have pain."

Her brother and neighbors stated that she was completely normal until two months prior to admission. At that time they noticed some peculiar behavior and forgetfulness. About two weeks before that she complained of deafness and dull pain in the left ear. Her referring physician removed some cerumen with a syringe. A few days later the ear began to drain. She then complained of increasing earache and radiating pain over the left eye and temple. For the last week, the drainage of the ear had stopped. Her headaches, however, became more pronounced. She became forgetful and mentally confused. She knew her brother but could not call his name.

*Physical examination* revealed a gracile, slightly stooped, pale patient. Temperature, 100.4° F.; blood pressure, 180/90, L.A.; 200/90, R.A; pulse, 80; respiration, 16.

*Eyes:* Chronic conjunctivitis and blepharitis; fundus, physiologic.

*Nose, Throat, Larynx:* Noncontributory.

*Neck:* Small, tender nodes at tip of mastoid behind tendon of mastosternocleido muscle.

*Ears:* Right, normal. Left, greenish fetid secretion in the canal. Large defect of the attic. Alcohol irrigation with Bezold attic cannula yielded foul-smelling masses of pus and debris from the epitympanic space. The left mastoid was tender to pressure, especially the areas of the emissary, auricular fold and linea temporalis.

*Nervus cochlearis* (see electroaudiogram, Fig. 2): Right ear, general defective hearing corresponding to age (presbyacusis). Left ear, complete deafness.

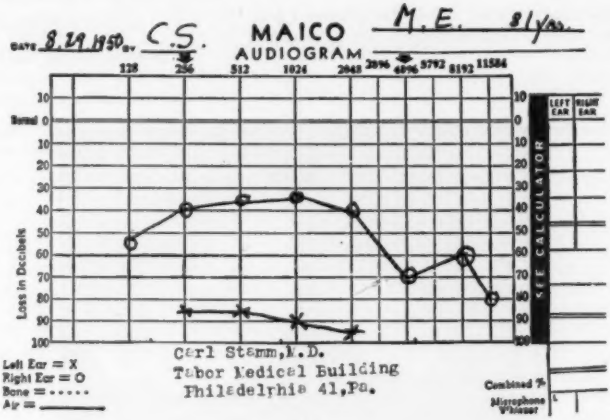


Fig. 2.

*Nervus Vestibularis:* No spontaneous nystagmus, no positive Romberg, no pastpointing, no fistula symptom, no response to irrigation with cold alcohol (nonfunctional labyrinth, left).

*Lungs:* Crepitant rales at the base bilaterally.

*Heart:* Blowing systolic murmur at the base.

*Abdomen and Extremities:* Not contributory.

*Nervous System:* Corneal sensation poor on both sides. No difference in objective sensation on the two sides. Loss of vibration sense in the legs. Positive biceps, negative triceps, negative plantar reflexes, poor patellar reflexes, negative Hoffmann, negative Babinski.

*Hematology:* Hgb., 10.5; R.B.C., 4.04 million; W.B.C., 7,800; poly., 62 per cent; mono., 1 per cent; lymph., 34 per cent; eosin., 2 per cent; fil., 96 per cent; nonfil., 4 per cent. Blood sugar, 93; B.U.N., 11; Wassermann, negative.

*Urinalysis:* Specific gravity, 1,007; pH, 4.5; no sugar, no abumin.

*Spinal Fluid:* Clear fluid, negative Pandy; pressure, 300 mm. of water.

*Roentgen Studies of Skull:* No shift of the pineal gland.

*Right Mastoid:* Normal. *Left Mastoid:* Sclerotic. There was a suggested area of bone destruction in the petrous pyramid. It was posterior and adjacent to the tegmen tympani (see Fig. 3).

*Diagnostic Impression:* Chronic destructive mastoiditis due to cholesteatoma could be established from the clinical findings. The labyrinth was nonfunctional. An osseous destruction near or adjacent to the dura of

the middle fossa was to be assumed according to the Roentgen findings. A brain abscess was a possibility because of the clinical symptoms (amnesic aphasia and mental disorientation, elevated spinal pressure).

*Operation Under Local Anesthesia* (Aug. 30, 1950): Postauricular incision, attic antromastoidectomy. Sclerotic contracted left mastoid was found and normal sigmoid sinus was exposed. An abscess filling an area

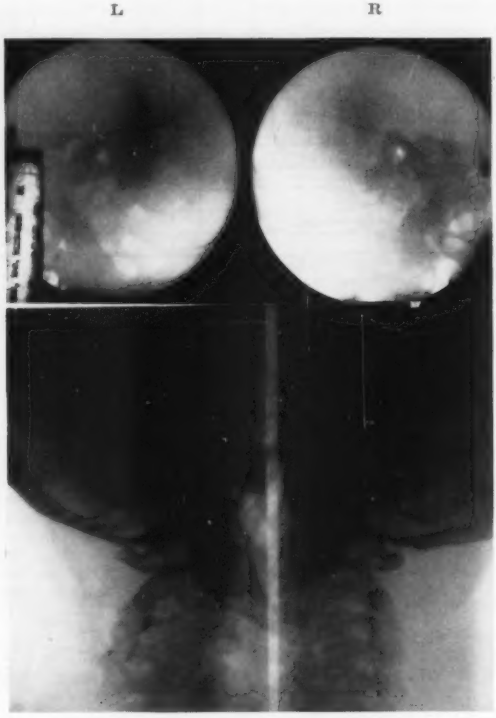


Fig. 3. Roentgen plates of mastoids.

of hazel nut size was opened near the antrum. It consisted of foul-smelling greenish-yellow pus, and covered the exposed dura, which was lined with thick granulations. In front of the abscess, cholesteomatous masses were found, filling large areas of the antrum, aditus ad antrum and the epitympanic recess. The incus was missing and the malleus was necrotic. Exposed and diseased dura was 0.5 cm. deeper than the roof of the antrum. The dura was pulsating. After removal of all necrotic bone, granulations and debris, the facial bridge was removed, and a door flap, formed out of the posterior cutaneous wall, was sutured to the anterior

aspect of the postauricular incision. A remnant of the tympanic membrane was mobilized from its lateral attachment and placed towards the opening of the Eustachian tube. The wound was kept wide open and packed with iodoform gauze.

*Laboratory Studies of Specimens:* Cholesteatoma, chronic osteitis, bacillus pyocyaneus, bacillus coli, staphylococcus aureus.

*Postoperative Course* (Sept. 8, 1950): No complaints of pain. Patient stated, "My mind seems to get clearer."

Sept. 18, 1950: The patient comprehended and expressed herself better. There was still some degree of nominal aphasia.

Sept. 28, 1950: Patient was discharged from the hospital. No trace of aphasia could be noted. Granulations of wound looked healthy.

May 21, 1951: The patient had been doing well. Postauricular fistula was kept open to control the exposed dura. It had not shown any signs of recrudescence; hence, the fistula was closed. The scars surrounding the fistula were excised, the cutaneous flap of the posterior auricular wall was dissected from its attachment, mobilized and sutured to the posterior aspect of the fistula. The fistula was closed with interrupted silk sutures after undercutting and mobilizing the periosteum of the mastoid.

April 30, 1952: Until this time, 20 months after the operation, the patient had not shown any signs of aphasic disturbances. The patient, now 83 years old, was completely alert. She had been keeping house for her widowed brother for over a year without the help of a maid.

## DISCUSSION.

An 81-year-old woman, who suddenly became disoriented, forgetful and amnesic aphasic, had complained for two weeks of severe left-sided earache and neuralgic pain over the left eye and left temple. A large cholesteatoma filled a defect of the epitympanic recess. The acoustic and static elements of the inner ear were dead. The mastoid area was tender to pressure. X-ray showed a decalcified area suggesting bone destruction in the petrous pyramid adjacent to the tegmen of the middle cranial fossa. Spinal pressure was elevated. All the data concerning history and clinical findings pointed to the diagnosis: chronic mastoiditis with intracranial spread of infection to the middle cranial fossa. A vital indication for a surgical intervention on the left temporal bone was given. In spite of this clear-cut syndrome, however, we watched the patient's condition for a week before we decided on the surgical therapy. An 81-year-old patient can present those psychiatric and aphasic symptoms without extensive destructive lesions of the brain tissue. That was proven by autopsies

performed by v. Monakow, Bianchi, Mingazzini, Lüers, Delay and Artom. They found only a cortical atrophy in the temporal lobe (Pick's disease).

The resident physician who admitted the patient had no information on the history. His first diagnostic consideration was, of course, focused to a vascular cerebral accident. This attitude was understandable. High blood pressure, loud systolic heart murmur and a hard, resisting pulse gave enough clinical evidence of general arteriosclerotic changes corresponding to the patient's age. An extensive intracranial hemorrhage could be excluded since the spinal fluid was clear. A few observations of amnesic aphasia of thrombovascular origin have been reported (Williams, Floris and L'Agostini). The majority of these lesions, however, will usually lead to a sensory aphasia of the Wernicke type. The patient's illness became manifest with the beginning of an amnesic aphasia. This is an important diagnostic sign pointing to an inflammatory lesion of the temporal lobe. In the true Wernicke aphasia due to vascular etiology the amnesia is the end-stage of the illness (Bonvicini).

It is difficult to explain the mechanism eliciting the aphasia and the general mental disturbances in our patient. We were not sure that the drainage of the extradural abscess was sufficient. Thormann's case similar to ours presented a temporal brain abscess one and one-half years later. Hinsberg saw aphasia disappear after radical mastoidectomy in two cases, but a few months later other signs of a brain abscess became manifest; thus, we took the expectant attitude, left the wound wide open and watched the exposed area for nine months until we closed the fistula. The release of the intracranial pressure is responsible for cure, especially in abscesses of large extension (Alexander, Price). Aphasia due to extradural hematoma (Ruttin, Scarff) and large hygroma (Sirois) explains this pressure mechanism *per se*. In our patient a relatively small abscess was found; we may, therefore, assume that a localized meningoencephalitis was responsible for the cerebral symptoms (Körner, Altmann, Ott, Eagleton). Others may incline more to the concept of Kurt Goldstein

that every aphasic patient shows changes pertaining to the personality as a whole. Price observed a peculiar misbehavior in children who were cured after exposure of a chronic extradural abscess of the temporal lobe. After the first World War, Goldstein, with the psychologist Gelb, studied a large number of soldiers who became aphasic after brain injuries. They concluded from their observations that from the circumscribed brain tissue primarily injured, the large score of psychopathological phenomena could be initiated. The primary lesion apparently operates like an electric switch, bringing the entire central system into upheaval. This is the revived idea of Pierre Marie, who in the beginning of this century stressed the importance of the psychopathology of the aphasic patient. These investigators were guided by a sentence spoken two generations before by the English neurologist, Hughlings Jackson: "To locate the damage which destroys speech and to locate speech are two different things." Aphasia was for Jackson in the first place a psychological problem: every aphasic patient, Jackson stated, descends from the highest levels of emotion and thoughts to the lower stages of regression and dissolution. It is worthwhile mentioning these facts in this connection because the modern psychological approach to therapeutic procedures in aphasia is based on this fundamental knowledge (Wepman, Goldstein, McBride and Weisenburg).

## SUMMARY.

*1.* A short description of types of aphasia occurring in otological practice is given.

*2.* An amnesic aphasia cured after exposure of an extradural abscess of the left temporal lobe is reported. The age of the patient (81 years) necessitated differential-diagnostic discussion.

*3.* Modern aspects on the psychopathological phenomena observed in aphasic patients were stressed.

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Tabor Medical Building.

# CHONDROMATOUS HAMARTOMA OF THE CRICOID CARTILAGE.*

YORK N. PITKIN, M.D., and DAVID M. SPENCER, M.D.,
Cleveland, Ohio.

Alexander,[1] writing in Berlin in 1900, classified cartilaginous tumors of the larynx into four groups: *1.* Chondromata which are true neoplasms and invade the mother substance from which they originate; *2.* mixed tumors such as chondrosarcoma; *3.* tumors of general hypertrophic or hyperplastic character affecting one or all of the cartilages of the larynx; *4.* inflammatory neoplasms; however, Alexander did not report any of his Type III tumor. New and Erich[2] reiterated this classification in 1938 in a review of 722 cases of benign tumors of the larynx. Again it was stated that none of the tumors classified in Group 3 had been seen by the authors. The case reported here essentially falls into Alexander's Group 3.

The term hamartoma has been used because it denotes hyperplastic development and connotates congenital existence. A cartilaginous hamartoma is not an unusual entity in itself; however, in a review of the literature of the past 20 years, there is no report of a chondroid hamartoma of the larynx under that or any other terminology. Reports of this type of tumor in the bronchi are not uncommon; therefore, it is more surprising that there is no previously recorded case of a laryngeal chondromatous hamartoma. Since the lesion is so unusual, it was felt it would be of sufficient interest to warrant presentation.

In an effort to establish an accurate terminology for this tumor, the sections made from the surgical specimen (see Fig. 1) were presented to three independent pathologists to-

*From the University Hospitals, Cleveland, Ohio.

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gether with the clinical picture. All three expressed the opinion that it was compatible with the diagnosis of chondromatous hamartoma. Age grouping and other statistical considerations, together with the pathologist's opinions, ruled out similar lesions in determining the terminology.

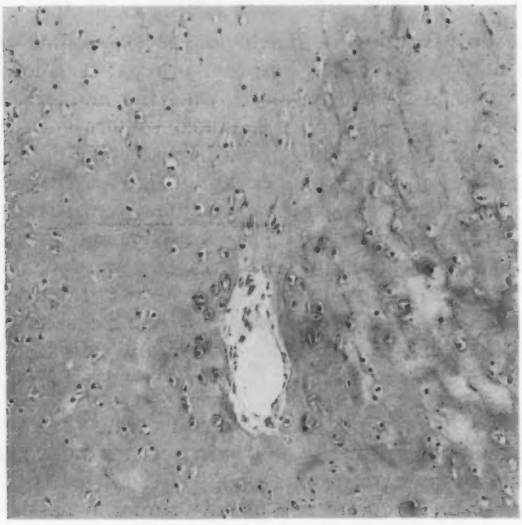

Fig. 1.

## CASE REPORT.

W. F., a Negro male, was first seen in the Pediatric Clinic at the Babies' and Children's Hospital of the University group on Oct. 3, 1951, at the age of 13 months. He was presented with the history of difficult breathing and hoarseness of three months' duration. According to the parents, prior to the onset of symptoms during July, 1951, he had been a normal healthy child. At this visit there was no indication of an inflammatory disease and he was sent to the Otolaryngological Clinic. He was seen there on Oct. 5 with inspiratory and expiratory stridor and croupy cry. No supraclavicular, suprasternal or intercostal retraction was noted. Direct laryngoscopy was scheduled and performed. This revealed swelling which appeared to arise from both sides of the subglottic portion of the larynx and to be bilaterally symmetrical and practically occluding the airway (see Fig. 2). The masses did not have the appearance of inflammatory disease, and the mucosa overlying did not have the prominent vascular supply and bluish coloring described as fairly characteristic of chondroma. No abnormality was palpable through the neck. The

tumors were firm to direct palpation and did not demonstrate pitting on pressure or react to the application of adrenalin. The child was placed on 100 mgm. of aureomycin every six hours for two weeks. At the end of this period, repeat laryngoscopy revealed that there was definitely no decrease in the size of the tumors, and, if anything, it was felt that the airway was even more embarrassed.

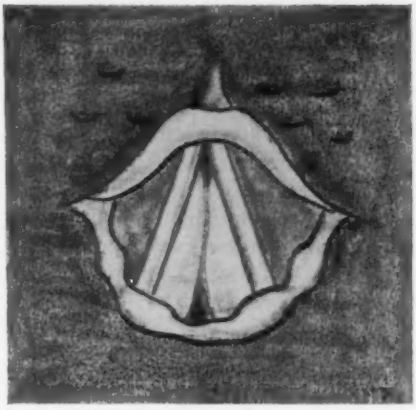

Fig. 2.

On Nov. 7, 1951, the patient was taken to the operating room and, under ether anesthesia, a tracheotomy was performed for intubation anesthesia and postoperative care. The cricoid was then split anteriorly and spread to expose the tumors. The mucosa and perichondrium were elevated from the surfaces of the two masses posteriorly to the midline, and all of the cricoid cartilage was resected, leaving the superior one-third of the posterior plate of the cricoid intact for the arytenoid articulation. The size of each mass measured approximately 1 x 0.5 x 0.4 cm. A tantalum tube was fixed in position with silver wire, and the mucoperichondrium was closed about it. Subcutaneous structures and skin were closed with silk. The immediate postoperative course was marred only by a minimal mediastinal emphysema which required no treatment.

Subsequent laryngoscopy, after the laryngeal edema subsided, showed that the arytenoids and vocal cords were functioning fairly well.

Laryngoscopy on Jan. 7, 1952, revealed considerable granulation tissue in and about the tantalum tube. The tube was removed on Jan. 16, 1952, through the tracheotomy wound and the granulations were cauterized with 10 per cent silver nitrate via the bronchoscope. Laryngoscopy and tracheoscopy was then performed at weekly intervals until the subglottic region was free of granulation tissue and appeared healthy.

After the granulation tissue had been controlled, dilatation was instituted. Metal dilators, size 18F through 22F, were passed at weekly intervals; however, there still was no cartilaginous support for the airway. It had been hoped that cartilage would reform from the perichondrium, which had been left in position in tubular form.

It is of interest to note that the child is developing well in other respects, playing with the other children in the ward, and now has a fairly good voice. The mother states that his voice at present is approximately the same as it was prior to surgery. There is not enough support to the airway to permit inspiration, but air can be forced past the tracheotomy tube in enough volume to produce vibration of the cords.

Attempts to implant endotracheal stents for support of the cricoid were repeatedly unsuccessful, and since there has been no evidence of stricture, it was decided to await the potential formation of cartilage while observing developments via the laryngoscope every two weeks. If cartilage fails to form, an anastomosis of the first tracheal ring with the thyroid cartilage must be considered.

In summary, we have presented an unusual lesion of the larynx, which we have classified as a chondromatous hamartoma of the cricoid cartilage, occurring in a 13-month-old Negro male. In a review of the literature for the past 20 years there has been no similar case reported. The end-result is not yet conclusive, and at present our course of action is simply observation at regular intervals.

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# PEDUNCULATED POLYP OF THE ESOPHAGUS ESOPHAGOSCOPIC REMOVAL.

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The successful removal of a myxomatous polyp from the esophagus is reported because *1.* the lesion is relatively infrequent;[1,2] *2.* to indicate that the average endoscopist, with no unusual instruments, can deal with this lesion.

## CASE REPORT.

Mrs. C. R., aged 33 years, was referred to me by Dr. Ralph D. Weible, internist, and Dr. Theodore L. Donat, radiologist, of the Dakota Clinic, Fargo, N. Dak., with a diagnosis of a pedunculated tumor of the esophagus. The patient complained of a lump in the throat. She said that occasionally something popped up into her throat and she could push it back with her finger and swallow it.

She had first noticed this sensation five years previously, and it had become worse. The patient had five children and was three months pregnant. She stated that her symptom was aggravated in the latter part of pregnancy. She complained of no difficulty in swallowing food nor did she have choking spells.

Physical examination was negative and mirror examination of the larynx and hypopharynx revealed no abnormalities.

Dr. Donat reported that there was hesitation at the beginning of the act of swallowing and the pyriform sinuses were not symmetrical. The epiglottis was normal. There was an area of fusiform dilatation approximately 6 cm. in the upper third of the esophagus. This area revealed a not completely fixed defect, and there was a suggestion of a pedicle at the lower portion of the dilated segment. *Impression:* polyp.

Esophagoscopy was performed under curare and pentothal anesthesia, with primary cocainization of the pharynx and larynx, using a short, adult Jesberg esophagoscope.

A short distance below the cricopharyngeus a sausage-like tumor 8 cm. long, not easy to differentiate from the wall of the esophagus, was identified. Because of its relative tenseness it could not be grasped with the ordinary flat grasping forceps. Right angled forceps were used to grasp its lower end and deliver it into the pharynx. The patient's breathing was immediately obstructed, and a bronchoscope was introduced as an airway.

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Fig. 1. Myxomatous polyp of the esophagus. Markedly reduced in size by fixation in alcohol.

Photo courtesy Dr. Frank M. Melton, Dakota Clinic, Fargo, N. Dak.

For a long time I have made a practice of having a suitable bronchoscope and introduction laryngoscope available when working with a patient under curare and pentothal, even during the introduction of an endolaryngeal catheter.

When the patient's airway had been established, the attachment of the polyp was found to be 2 to 3 cm. below the cricopharyngeus on the lateral wall of the esophagus. Because the attachment was not particularly thin, it was felt that electrocoagulation was not justified. Simple snaring was deemed the safest method of removal. To be prepared for this, the obturator of one of the larger endoscopic forceps had been replaced by a loop of ordinary tonsil snare wire, the ends of which were controlled by a hemostat. This snare was passed over the polyp, care being taken not to use excessive tension on the polyp so that the wall of the esophagus would not be included in the loop. The polyp was removed. It was apparent that the mucosa alone, and not the body of the polyp itself, had been severed in an area of 1.5 cm.

There was not much bleeding. The patient was given penicillin for five days and kept on a liquid diet for 10 days. Convalescence was entirely uneventful, and the patient has had no more symptoms.

The patient was delivered at term, March 6, 1952.

Fluoroscopic examination of the esophagus on July 7, 1952, revealed no abnormality.

Pathological report by Dr. J. J. Spier, pathologist of St. John's Hospital, Fargo, N. Dak.: "A plum-shaped portion of rubbery, grayish tissue, measuring 5.5 by 3 by 2.5 cm. On one end it tapers off to quite a point. On section it appears to be well encapsulated and consists of soft, myxomatous, grayish tissue." Note that this description as well as the picture illustrating the polyp were after fixation in alcohol. At the time of removal the tumor was much larger.

"Sections show a tumor which is covered by stratified squamous epithelium. The tumor itself is composed of a loose matrix containing frayed out coarse, collagenous fibres and a mucoid coagulum. The cells, especially in some fields, are stellate or spider-like. There is some vascularity and scattered round cell infiltration. One cannot recognize muscular elements of known types.

"Diagnosis: Myxomatous polyp."

## CONCLUSION.

It is occasionally possible for the endoscopist to remove a pedunculated, benign tumor of the esophagus completely by a procedure which is much simpler than transthoracic esophagotomy.

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2. Harrington, S. W.: *Arch. Surg.*, 58:646, May, 1949.

15 Broadway.

# A MODIFIED TECHNIQUE OF GENERAL ANESTHESIA FOR LARYNGECTOMY.*†

B. I. SCHNEIDERMAN, M.D., and S. L. PERZIK, M.D.,
Beverly Hills, Calif.

Numerous methods have been devised for the administration of general anesthesia during laryngectomy. Of these, the one most frequently employed utilizes an oroendotracheal catheter[2,3] during the first part of the procedure, while the larynx is skeletonized. After the larynx is freed and the trachea is transected, the oroendotracheal tube is removed and a clean one is inserted through the tracheal opening by the surgeon who passes the free end underneath the drapes to the anesthetist for the maintenance of anesthesia. In those instances where a tracheostomy already exists, the agents may be introduced directly through it, and for this purpose special adaptors have been devised by Sanders,[5] among others, which attach directly to the tracheostomy tube or are inserted through it. Others use shortened endotracheal catheters,[3] if the existing tracheostomy is large enough to permit their insertion. Some maintain anesthesia with pentothal sodium while administering oxygen by means of a urethral catheter inserted through the tracheotomy tube.

There are numerous difficulties and inconveniences inherent in these methods. When the two-catheter technique is employed, it may be technically impossible to insert the endotracheal catheter past the tumor mass, or part of the mass may be broken off. Since, in any event, the patient is to end up with a tracheostomy, the expenditure of time and effort to introduce the tube orally, and the further delay associated with the reintroduction of the second endotracheal tube through the tracheal stoma is both wasteful and unwarranted.

*Presented at the California Medical Association Meeting at San Diego, Calif., April 29-May 3, 1950.

†From the Department of Surgery (Subsection of Anesthesia and Subsection of Head and Neck), Cedars of Lebanon Hospital, Los Angeles, Calif.

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In addition, when the operation is near termination and only the tracheal skin anastomosis remains to be performed, the surgeon is still faced with the handicap of contending with the endotracheal tube protruding from the cut end of the trachea. Should a tracheostomy already exist, due to a pre-existing respiratory obstruction, the surgeon is then faced with the responsibility of maintaining a free airway and contending with blood and secretions which may spill down into the trachea and bronchi when a tracheotomy adaptor or urethral catheter is used.

In order to overcome the preceding difficulties and to simplify the procedure, the following technique was devised: Under pentothal anesthesia and local infiltration with 1 per cent procaine, the initial step in the entire operation consists of a longitudinal tracheotomy incision below the point of the proposed transection of the trachea. In the presence of a previous tracheostomy, it can be made as low as the sixth tracheal ring. Extreme extension of the neck facilitates this maneuver. Prior to the actual tracheotomy incision, in order to prevent laryngospasm, bucking and the cardiac irregularities that are sometimes associated with intubation, the surgeon injects 1 cc. of 10 per cent cocaine or 2 per cent pontocaine through the site of the proposed incision. After the tracheotomy is made, a latex-covered wire-wound endotracheal tube, 32-36 French, with an inflatable cuff, which has previously been sterilized in alcohol or other suitable agent, is inserted through the tracheal slit by the surgeon. The insertion can be facilitated by using a tracheotomy spreader. The tube is then anchored in position by a heavy silk suture passed through the skin and tied tightly around the tube. If difficulty in free exchange is noted, the tube must be checked to be certain that it has not been pulled out or pushed in too far, or that its beveled face is not in flat contact with the wall of the trachea. The wire-wound tube is utilized because of its flexibility and the fact that it cannot collapse even when bent at extreme angles. The cuff is then inflated, and the free end of the tube is passed caudad beneath the drapes to the anesthetist who may maintain anesthesia with any agent or method he desires (see Fig. 1).

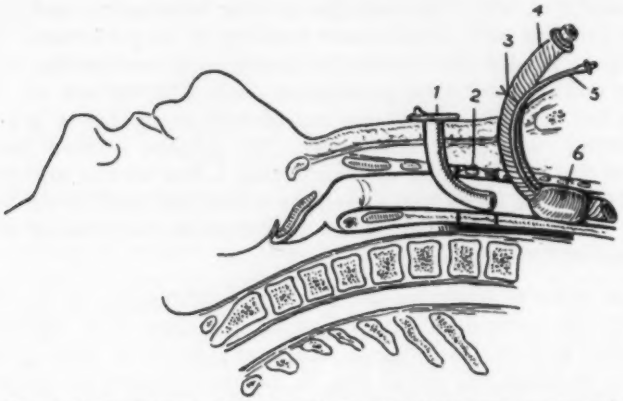


Fig. 1. (1) Pre-existing tracheotomy. (2) Site of proposed section of trachea. (3) Silk suture anchoring latex-covered wire-wound endotracheal catheter (4). (5) Inflating tube. (6) Cuff.

The surgeon can now perform and complete the entire operation without the necessity of changing the anesthetic set-up. After transection of the trachea, the surgeon has free access to the stoma while making the skin-tracheal anastomosis, as the endotracheal tube is through a slit well below this point (see Fig. 2). Upon completion of the operation, the endotra-

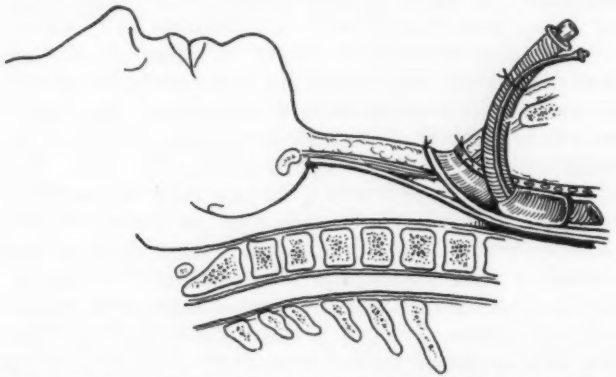

Fig. 2. The completed permanent skin-tracheal anastomosis above the temporary tracheotomy through which the cuffed endotracheal tube was initially passed.

cheal tube is removed and the corresponding tracheal slit is permitted to close spontaneously. This area is drained for 24 hours.

This procedure has now been used in 24 cases of various types of laryngectomy and has proved completely satisfactory regardless of the extent and location of the pathology encountered. Ten of the cases were simple laryngectomies with free airways preoperatively. Nine other operations involved the removal of the larynx with part of the tongue, hyoid bone, pre-epiglottic space, strap muscles and thyroid. Eleven of these cases had preoperative tracheostomy tubes in place because of obstructed airways, a condition which would have made the passage of a naso- or oroendotracheal tube impossible. In three cases, a laryngectomy was combined with a unilateral radical neck dissection and in two instances a laryngoglossectomy was combined with a simultaneous bilateral radical neck dissection.

## SUMMARY.

A modified technique to be used with general anesthesia in laryngectomy is described.

It permits the entire operation to be completed from the very first step of the procedure to its termination without the necessity of changing the anesthetic set-up.

It facilitates the performance of the skin-tracheal anastomosis.

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# In Memoriam

JOHN J. SHEA, M.D., 1889-1952.

It is our sad duty to record the passing of another member of our editorial staff, Dr. John J. Shea, of Memphis, Tenn., on Nov. 28, 1952.

Dr. Shea was born in Memphis on Jan. 17, 1889, the son of Joseph and Mary Coyle Shea. He attended school in Memphis, and in 1906 he was granted a B.A. from Christian Brothers College. In June, 1911, he received his M.D. degree from New York University and Bellevue Medical College. His interneships were served at St. John's Seaside Hospital, Staten Island, Manhattan Maternity Hospital, and Bellevue Hospital Third Medical Division, and he also served as resident in that service during 1913. During 1914-1915 he did post-graduate work at New York Post-Graduate Hospital and in 1918-1919 he studied at the University of Paris.

In 1914 he became associated with Dr. Ellett and Dr. Farrington, of Memphis.

During World War I, Dr. Shea was made a first lieutenant in 1917, captain in 1918, and major in 1919, serving with the Medical Section of Aviation Corps; Base Hospital, Camp Meade; Red Cross Hospital No. 7, Juilly Seine et Marne; Base Hospital No. 57, AEF, Paris, France. He was also examiner for the Department of Aeronautics from 1925 through 1932.

Dr. Shea was Visiting Otolaryngologist at Baptist Memorial, St. Joseph's, and Memphis Eye, Ear, Nose and Throat Hospital; he was Consultant for Campbell Clinic and for Kennedy General Hospital.

He was a Fellow of the American Medical Association, and was secretary of the Section on Otolaryngology in 1931-1932-1933, serving as its chairman in 1934-1935; Southern Medical Association, serving as its secretary of the Section on Ophthalmology and Otolaryngology, 1920-1921-1922, vice-chairman in 1923, and chairman in 1924; the American College of Surgeons, 1922; the American Academy of Ophthalmology and Otolaryngology, serving on the council in 1929-1930-1931-1932, as vice-president in 1924, and was president-elect for 1953. He was president of the American Laryngological, Rhinological and Otological Society in 1948-1949, and was a member of the Council in 1937-1938-1939. In 1948 and 1949 he served as president of the American Otological Society. In 1944-1945 and 1947, he was first vice-president of the American Laryngological Association.

On July 5, 1917, Dr. Shea was married to Miss Catharine Flanagan and they had six children.

In addition to his contributions to his specialty through his society and hospital affiliations, Dr. Shea had contributed many papers to otolaryngologic literature.

Dr. Shea considered the practice of medicine as a great privilege and he prepared himself for this responsibility by careful study and unstinted service, yet he was always the humble and understanding type. He was kind, gentle and loyal and for these attributes he will long be remembered by his friends, patients and colleagues.

## AMERICAN HEARING SOCIETY OPENS ANNUAL COMPETITION — KENFIELD MEMORIAL AWARD.

American Hearing Society announces March 1 as opening date of competition for the 1953 Kenfield Memorial Scholarship awarded annually by this agency to a prospective teacher of lipreading to the hard of hearing. Application blanks may be obtained from Miss Rose V. Feilbach, Chairman of the Society's Teachers Committee, 1157 North Columbus Street, Arlington, Va. Deadline for returning applications, completed, is May 1.

Winner of the award is entitled to take a teacher training course in lipreading from any normal training teacher, school or university in the United States acceptable to the American Hearing Society's Teachers Committee. The scholarship is to be used within one year from date of award.

An acceptable applicant for the scholarship must be a well adjusted individual with a pleasing personality, legible lips, a good speech pattern and no unpleasant mannerisms. Graduation from college with a major in education, psychology and/or speech is a requirement. Specifications for a hard-of-hearing contestant include 30 clock hours of private instruction in lipreading from an approved teacher or 60 hours of lipreading in public school classes under an approved teacher. Rules for competition state that an applicant shall plan to teach lipreading, with or without other types of speech or hearing therapy.

## XVIII CONGRESSO INTERNAZIONALE OTO-NEURO-OFTALMOLOGICO.

The XVIII International Congress of Oto-Neuro-Ophthalmology will be held in Bologna, Italy, May 3-7, 1953. Chairman: Prof. Q. Di Marzio. Official reports are:

*1. Dysrraphia in Oto-Neuro-Ophthalmology:* General Reporter: Belloni, Padua. Embryology: Krabbe, Köbenhavn. Neurology: Vercelli, Milan. Radio-Neurology: Mascherpa, Milan. Oto-Laryngology: Arslan, Padua. Ophthalmology: Franceschetti, Geneva. Neuro-Surgery: Fasiani, Milan. Facial-Surgery: Sanvenero-Rosselli, Milan.

*2. Ménière's Disease:* Reporters—Neurology: Barré, Strasbourg. Otology: Greiner, Strasbourg. Neuro-Surgery: Tolosa, Barcelona.

The official languages admitted are: Italian, English, German, French, Spanish.

Besides the above mentioned subjects, papers will be also accepted on arguments regarding at least two of the specialties at the same time.

For further information address Dr. Giuseppe Cristini, Clinica Oculistica-Policlinico, Bologna, Italy.

## HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE OF THE AMERICAN MEDICAL ASSOCIATION.

January 1, 1953.

Audicon Models 400 and 415.
Manufacturer: National Earphone Co., Inc., 20-22 Shipman St., Newark 2, N. J.

Auditone Model 11.
Manufacturer: Audio Co. of America, 5305 N. Sixth St., Phoenix, Ariz.

Audivox Model Super 67.
Manufacturer: Audivox, Inc., 259 W. 14th St., New York 11, N. Y.

Aurex Models L and M.
Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago, Ill.

Beltone Symphonette; Beltone Mono-Pac Model M.
Manufacturer: Beltone Hearing Aid Co., 1450 W. 19th St., Chicago, Ill.

Cleartone Model 500; Model 700; Cleartone Regency Model.
Manufacturer: American Sound Products, Inc., 1303 S. Michigan Ave., Chicago 5, Ill.

Dahlberg Model D-1; Dahlberg Junior Model D-2; Dahlberg Model D-3; Dahlberg Model D-4.
Manufacturer: The Dahlberg Co., Golden Valley, Minneapolis 22, Minn.

Dysonic Model 1.
Manufacturer: Dynamic Hearing Aids, 149 Church St., New York 7, N. Y.

Electroear Model C.
Manufacturer: American Earphone Co., Inc., 10 East 43rd St., New York 17, N. Y.

Gem Hearing Aid Model V-35; Gem Model V-60.
Manufacturer: Gem Ear Phone Co., Inc., 50 W. 29th St., New York 1, N. Y.

Goldentone Models 25, 69 and 97.
Manufacturer: Johnston Hearing Aid Mfg. Co., 708 W. 40th St., Minneapolis 8, Minn.
Distributor: Goldentone Corp., 708 W. 40th St., Minneapolis 8, Minn.

Maico UE-Atomeer; Maico Quiet Ear Models G and H; Maico Model J.

Manufacturer: Maico Co., Inc., 21 North Third St., Minneapolis 1, Minn.

Mears (Crystal and Magnetic) Aurophone Model 200; 1947—Mears Aurophone Model 98.

Manufacturer: Mears Radio Hearing Device Corp., 1 W. 34th St., New York, N. Y.

Micronic Model 303; Micronic Model "Mercury"; Micronic Star Model.

Manufacturer: Audivox, Inc., Successor to Western Electric Hearing Aid Division, 123 Worcester St., Boston 18, Mass.

Microtone T5 Audiomatic; Microtone Classic Model T9; Microtone Model T10; Microtone Model T612; Microtone Model 45.

Manufacturer: Microtone Co., Ford Parkway on the Mississippi, St. Paul, Minn.; Minneapolis 9, Minn.

National Cub Model C; National Cub Model D (Duplex); National Standard Model T; National Star Model S; National Ultrathin Model 504; National Vanity Model 506.

Manufacturer: National Hearing Aid Laboratories, 815 S. Hill St., Los Angeles 14, Calif.

Normatone Model C.

Manufacturer: Johnston Hearing Aid Mfg. Co., 708 W. 40 St., Minneapolis, Minn.

Distributor: Normatone Hearing Aid Co.

Otarion Model E-4; Otarion Models F-1, F-2 and F-3; Otarion Model G-2; Otarion Model G-3.

Manufacturer: Otarion Hearing Aids, 4757 N. Ravenwood, Chicago 40, Ill.

Paravox Model D, "Top-Twin-Tone"; Model J (Tiny-Mite); Paravox Model XTS (Xtra-Thin); Paravox Model Y (YM, YC and YC-7) (Veri-Small).

Manufacturer: Paravox, Inc., 2056 E. 4th St., Cleveland, Ohio.

Radioear Permo-Magnetic Multipower; Radioear Permo-Magnetic Uniphone; Radio Ear All Magnetic Model 55; Radioear Model 62 Starlet; Model 72.

Manufacturer: E. A. Myers & Sons, 306 Beverly Rd., Mt. Lebanon, Pittsburgh, Pa.

Distributor: Radioear Corp.

Rochester Model R-1; Rochester Model R-2.

Manufacturer: Rochester Acoustical Laboratories, Inc., 117 Fourth St., S.W., Rochester, Minn.

Silvertone Model J-92; Silvertone Model P-15.

Manufacturer: W. E. Johnson Mfg. Co., 708 W. 40th St., Minneapolis, Minn.

Distributor: Sears, Roebuck & Co., 925 S. Homan Ave., Chicago 7, Ill.

Solo-Pak Model 99.

Manufacturer: Solo-Pak Electronics Corp., Linden St., Reading, Mass.

Sonotone Model 900; Sonotone Models 910 and 920; Sonotone Model 925; Sonotone Model 940; Sonotone Model 966.

Manufacturer: Sonotone Corp., Elmsford, N. Y.

Superfonic Hearing Aid.

Manufacturer: American Sound Products, Inc., 1303 S. Michigan Ave., Chicago 5, Ill.

Televox Model E.

Manufacturer: Televox Mfg. Co., 1307 Sansom St., Philadelphia 7, Pa.

Telex Model 97; Telex Model 99; Telex Model 200; Telex Model 300B; Telex Model 400; Telex Model 500; Telex Model 952; Telex Model 1700.

Manufacturer: Telex, Inc., Telex Park, St. Paul 1, Minn.

Tonamic Model 50.

Manufacturer: Tonamic, Inc., 12 Russell St., Everett 49, Mass.

Tonemaster Model Royal; Model Cameo.

Manufacturer: Tonemasters, Inc., 400 S. Washington St., Peoria 2, Ill

Unex Midget Model 95; Unex Midget Model 110; Unex Models 200 and 230.
Manufacturer: Nichols & Clark, Hathorne, Mass.

Vacolite Models J and J-2.
Manufacturer: Vacolite Co., 3003 N. Henderson St., Dallas 6, Tex.

Western Electric Models 65 and 66.
Manufacturer: Audivox, Inc., Successor to Western Electric Hearing Aid Division, 123 Worcester St., Boston 18, Mass.

Zenith Miniature 75; Zenith Model Royal; Zenith Model Super Royal.
Manufacturer: Zenith Radio Corp., 6001 Dickens Ave., Chicago, Ill.

All of the accepted hearing devices employ vacuum tubes.

Accepted Hearing Aids more than five years old have been omitted from this list for brevity.

## TABLE HEARING AIDS.

Ambco Hearing Amplifier (Table Model).
Manufacturer: A. M. Brooks Co., 64 S. Bonnie Brae St., Los Angeles 5, Calif.

Aurex (Semi-Portable).
Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago 10, Ill.

Precision Table Hearing Aid.
Manufacturer: Precision Hearing Aids, 5157 W. Grand Ave., Chicago 39, Ill.

Sonotone Professional Table Set Model 50.
Manufacturer: Sonotone Corp., Elmsford, N. Y.

All of the Accepted hearing devices employ vacuum tubes.

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Time and Place: January, 1954, Mexico City.

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Meeting:

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Meeting: Hot Springs, Va., May 2-3, 1953.

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Secretary: Dr. W. Ross Wright, 361 Regent St., Fredericton, N. B.
Place: Minaki Lodge, Minaki, Ontario.
Time: June 14-18, 1953.

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